Journal OF THE AMERICAN VETERINARY MEDICAL ASSOCIATION

Joint Pan American-AVMA Meeting, Kansas City, August 23-27, 1959

General Articles

Surgery and Obstetrics

Clinical Data

Editorial

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Vol. 134 June 1, 1959 No. 11

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Journal of the American Veterinary Medical Association

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Correspondence

May 1, 1959

Dear Dr. Aitken:

Your review of the book "Diseases of Swine" has just crossed my desk and I am pleased with the fine things which you said about it. It was hoped that it would be as complete as possible for the variety of readers who would be interested in swine diseases. It is recognized that, in this broad coverage, some areas may have been treated too lightly. As these points are brought to our attention, we will attempt to emphasize their correction in the first revision.

I feel that it is necessary, however, to defend the statement in the book to which you have taken exception. You said "to state 'that current infection with another septicemic organism such as *Erysipelothrix rhusiopathiae* is a common cause of so-called serum breaks' is just not true." The statement in the book reads, "the most common factor (for serum breaks) is the simultaneous infection with another septicemic organism such as *Erysipelothrix rhusiopathiae, Salmonella cholera suis, Pasteurella multocida,* or streptococci." Other organisms likewise may be involved in a simultaneous assault upon the animal's defense mechanism.

1) First of all, *Erysipelothrix rhusiopathiae* was not singled out as "the common cause" and was indicated only as one of many organisms which can cause septicemic invasion, and thus contribute to the "common cause" of serum breaks.

2) You state that, "it was routine procedure to treat nonvaccinated herds in which some pigs were sick with acute erysipelas with regular doses of anti-hog cholera serum and virulent virus, and also to give visibly sick pigs a dose of penicillin or anti-swine erysipelas serum." Most veterinarians with whom I have worked invariably gave somewhat increased doses of anti-hog cholera serum plus the additional treatment suggested when the presence of another disease was anticipated. Cases in which the swine erysipelas organism was found in hog cholera "breaks," in my experience, were most frequently observed in those cases where the serum dosage could not have been considered "generous."

3) That there is a considerable swine erysipelas antibody content in the average midwestern-produced anti-hog cholera serum is recognized. But to assume that all serum produced under these conditions is high in anti-swine erysipelas antibody is likewise a questionable presumption. In my own experience with the testing of anti-hog cholera serum, the content of swine erysipelas antibodies in most lots was high. However, some lots were relatively low.

4) No intelligent research man would underestimate the diagnostic capabilities of a good practitioner. But we who are working with diseases under experimental conditions are amazed at the occasional misinterpretation made upon clinical observations alone. I'm sure that the progressive practitioner appreciates the increased accuracy of diagnosis which is provided by the laboratory in addition to his clinical observations. Therefore, I wonder if one can lump selected clinical observations into a compact package, and state that the excerpt from the book "is just not true." I would think that a complete review of all of the records which you have kept, including any "breaks" which you may have had, that were not explained, should be carefully analyzed before such a flat statement could be made.

I seriously question whether the inclusion of *Erysipelothrix rhusiopathiae* with the group of organisms mentioned as a common factor in "serum breaks" can be classified as "a great committed error."

We do, however, welcome such criticism for it gives us the opportunity to strengthen the weak spots in the publication. Actually we are not far apart in our thinking and you can be assured that the anti-swine erysipelas potential of anti-hog cholera serum will be given full attention in the revision of the book.

Again, many thinks for a fine review.

Sincerely yours,

s/H. W. DUNNE, D.V.M., Ph.D.

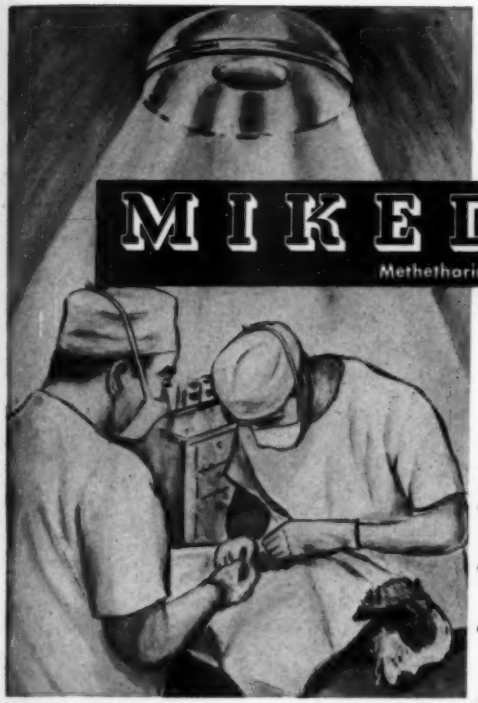
University Park, Pa.

[Dr. Dunne says, "Actually we are not far apart in our thinking . . . ." — I agree. Our difference is in the approach. His is chiefly from the research laboratory; mine was from the field (JOURNAL, Jan., 1950: 41). This vital difference is one which younger veterinarians may not know about but which will not be forgotten by those who were involved. It is that swine erysipelas was a major swine killer, especially in the northwestern tip of the corn belt, for over 20 years before research workers in America were able to contribute much help to those on the firing line, except by diagnoses. Why? Because erysipelas could seldom be reproduced for study under controlled conditions. Thus, the immunity resulting from vaccination could not be measured by challenge and the cause of immunizing failures (breaks) could not be determined. For that reason, field research with swine erysipelas became an intriguing challenge.

Points on which Dr. Dunne and I agree (and the qualifications):

1) Septicemic infections, and chronic infections, frequently cause tragic complications when present in pigs during the reaction period which follows vaccination with either virulent or modified hog cholera virus vaccines. (However, this is surprisingly seldom true with erysipelas. I'm positive it did not occur in my practice.)

2) Pigs should be given "generous" doses of serum in the presence of other disease. (My



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"regular" doses of 30 cc. to 30-lb. pigs and 60 cc. to 100-lb. shoats were equal to the "increased" doses of many. The "increase" was good insurance for the pig crop and for sounder sleeping by the owner and vaccinator.)

3) There are considerable but variable quantities of erysipelas antibodies in anti-hog cholera serum. (They should be most plentiful in serum from hogs raised where erysipelas is most prevalent.)

4) Practitioners should appreciate the "increased accuracy of diagnosis . . . provided by the laboratory." (I would not have dared, over the years, to express so many opinions on erysipelas which were contrary to those then held by "officials" and "authorities" had I not, for 20 years, kept detailed "records" on all cases and "breaks" and had not my field diagnoses usually been confirmed in a well-qualified laboratory. It is gratifying and surprising to learn how well every detail of those field observations stood up after laboratory research became possible.)

Swine erysipelas has generally been considered an unusually unpredictable disease, largely because it frequently does not conform to established principles or theories which apply to other infectious diseases. Thus, differences of opinions are common.

Again referring to the erysipelas antibodies in anti-hog cholera serum, we can expect the incidence of erysipelas to increase as the use of large doses of this serum decreases. Why? Because the peak season for cholera vaccinating (May and June) just precedes the peak season for acute erysipelas (June and July), and the pigs given large doses of serum are thus protected for a few weeks at the time they are most apt to be exposed to erysipelas. Presumably, the antibodies plus natural exposure result in considerable active immunity.

The antibody content also explains why good immunity against erysipelas should not be expected when pigs are vaccinated simultaneously against erysipelas and cholera, when anti-hog cholera serum is used (see p. 497).—W. A. AITKEN.]

Instructions to Authors

JOURNAL *of the AVMA*

Exclusive Publication.—Articles submitted for publication are accepted with the understanding that they are not submitted to other journals, which is ethical publication procedure.

Manuscripts.—Manuscripts, including **footnotes, references,** and **tables,** must be typewritten, **double-spaced,** on 8½- by 11-in. bond paper, and the original and one carbon copy, submitted. One-inch margins should be allowed on the sides, with 2 in. at top and bottom. Articles should be concise. Short, simple sentences are clearer and more forceful than long, complex ones.

Illustrations.—Photographs should be furnished in glossy prints, and of a size that will fit into the JOURNAL of the American Veterinary Medical Association with a minimum of reduction. Photomicrographs which can not be reduced should be marked for cropping to 1-column or 2-column width. Identifying marks within the photomicrographs, such as arrows, letters, or numbers, should be clearly marked with black India ink or white opaque ink to insure good contrast for reproduction and must be large enough to stand reduction, if necessary.

Drawings, graphs, and charts should be made clearly and accurately in India ink on white paper and a glossy print of them submitted when possible. Numbers or letters appearing on graphs or charts should be large enough to allow for any reduction necessary for the chart or graph to fit JOURNAL pages. Blue lines in graph paper drop out in reproduction; therefore, if lines are required they must be drawn in black ink. All illustrations should bear the name of the author and the illustration number on the back.

Tables.—Tables should be simple and typed **double space.** Complex tables are not conducive to perusal. It is wiser to summarize complex material than to tabulate it.

References.—References should be typed **double space,** in alphabetical order, and should be prepared in the following style: name of author, title of article, name of periodical with volume, year, and page numbers. References to journals not commonly known should give the complete name of the periodical, and where published so that they may be added to our reference files. When books are cited, the name of publisher, location, edition, and year should be given.

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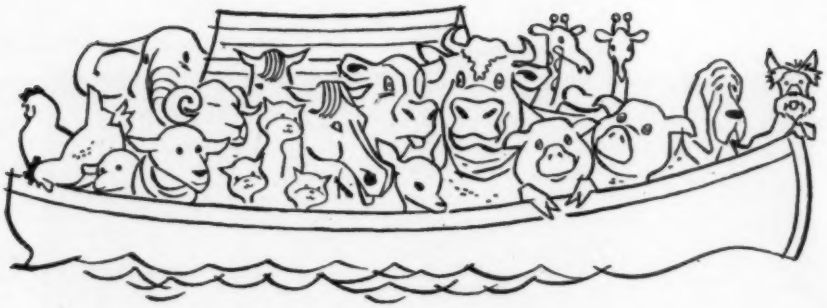

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1. Barr, F.S., Carman, P.E. and Harris, J.R.: Synergism and Antagonism in Antibiotic Combinations; Antibiotics and Chemotherapy; 4:818 (1954).
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3. Barr, F.S., Harris, J.R. and Carman, P.E.: Intramuscular Treatment of Staphylococcic Mastitis with Neomycin Sulfate and Polymyxin B Sulfate; J.A.V.M.A., 132 (1958): 110.

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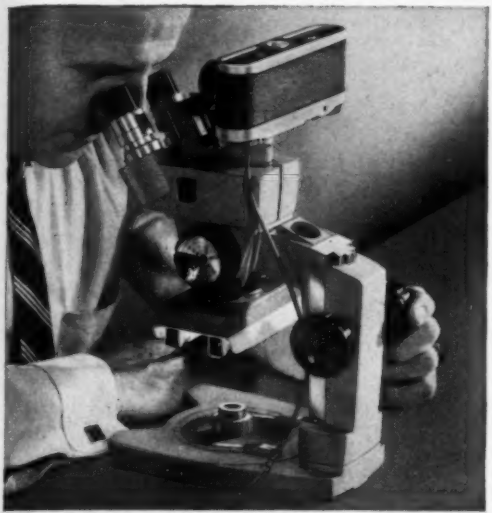

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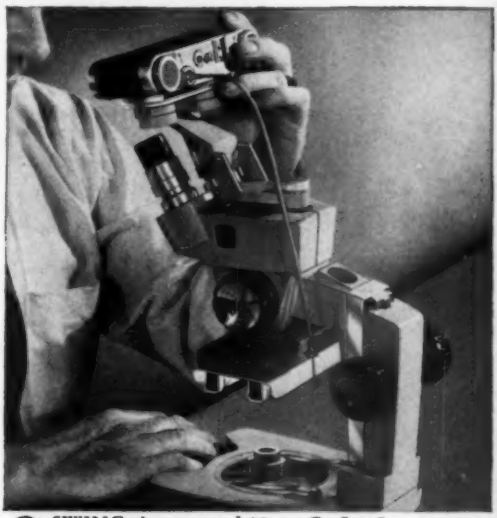

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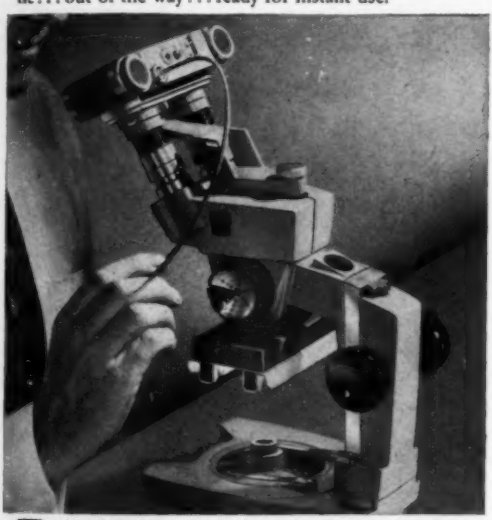

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Washington News

Legislative

Senate Committee on Appropriations, when considering the **Second Supplemental Appropriations Bill** (H.R. 5916) for 1959, recommended that $100,000 be included to **initiate research on humane slaughter methods,** with a maximum of $35,000 to be allocated for alterations to the meat laboratory at Beltsville, Maryland. The House had denied the budget request, suggesting the item be considered in the regular appropriations bill for the fiscal year 1960. The Senate committee believes that the responsibilities imposed upon the Secretary of Agriculture in carrying out the research work required by Public Law 85-765 (Humane Slaughter Act) should not be delayed. This committee also recommended that the Food and Drug Administration receive the $378,000 requested in the supplemental budget to finance initial implementation of the Food Additives Amendment of 1958, which the House had reduced by $78,000.

H.R. 5916, with amendments, passed the Senate, April 30, 1959. Bill now goes to Senate-House conference. Senate Labor and Public Welfare Committee reported favorably **S.J.Res. 41,** authorizing $50 million annually to foster international medical research (see JOURNAL, March 15, 1959, adv. p. 16). Early Senate passage is expected.

H.R. 4012, commemorating the centennial of the establishment of land-grant colleges and state universities and of the Department of Agriculture, as amended, passed the House, April 20, 1959 (see JOURNAL, March 15, 1959, adv. p. 16). One amendment authorized appropriations not to exceed $200,000. The Senate Finance Committee, may, according to reports, schedule hearings on **H.R. 10 (Keogh-Simpson tax deferment)** in May or June of this year (see JOURNAL, April 15, 1959, adv. p. 16).

New Bills

Following are new bills to amend Title II, Social Security Act.

H.R. 6513, Rep. Broomfield (R.,Mich.), would make age 60 the retirement age for old age and survivors insurance benefits. **H.R. 6520,** Rep. Flynn (D.,Wis.), increase all benefits by ten per cent and provide full benefits at age 60. **H.R. 6538,** Rep. Rogers (D.,Fla.), to increase from $1,200 to $3,600 the amount of outside earnings permitted without deductions from benefits.

H.R. 6421, Rep. Foley (D., Md.), to establish a system for the classification and compensation of scientific and professional positions in the federal government.

H.R. 6436, Rep. Cooley (D., N. Car.), to amend the Federal Insecticide, Fungicide, and Rodenticide Act to include nematocides, plant regulation, defoliants, and desiccants.

H.R. 6595, Rep. Abbitt (D., Va.), to amend Title III, Packers and Stockyards Act of 1921, with respect to regulation of small stockyards under that title.

S. 1742, Sen. Magnuson (D., Wash.), to amend Federal Trade Commission Act to prohibit certain practices in commerce by any manufacturer or producer who distributes his product through his own retail outlets, direct to consumers, and also through other retail outlets.

S. 1683, Sen. McCarthy (D., Minn.), to amend Hatch Act, to permit all officers and employees of the Federal government to exercise full responsibility of citizenship and take an active part in the political life of the United States.

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Washington News—*Continued*

S. 1711. Sen. Humphrey (D., Minn.), and 15 co-sponsors, to promote the foreign policy of the United States and help build essential world conditions of peace by more effective use of U.S. agricultural commodities for relief of human hunger, and for promoting economic and social development in less developed countries. The bill, in amending Public Law 480, 83rd Congress, would include amendments to sections relating to international educational exchange activities, and scientific activities. The latter amendment reads: ". . . and to promote and support programs of medical and scientific research, cultural and educational development, health, nutrition, and sanitation."

H.Con.Res.113, Rep. Johnson (D.,Col.), providing for the development through United Nations of international educational programs.

U.S.D.A.

Notice in "Federal Register," April 22, 1959, further **amends applicable federal regulations which restricts the interstate movement of cattle and sheep because of scabies.** The primary purpose of the several amendments, effective April 22, 1959, is **to remove benzene hexachloride from the list of dips permitted** by the Department of Agriculture for the treatment, under division supervision, of cattle and sheep affected with or exposed to scabies. **It also reflects limitations on the use of the permitted lindane dip.**

International Animal Feed Symposium, sponsored by U.S.D.A. and the Soybean Council of America, Inc., was held in Washington, D.C., May 4-6, 1959. The purpose of the symposium was designed **to encourage greater use of United States feedstuffs in Europe.** About 30 specialists from ten Western European countries, including specialists in feeding and animal nutrition, joined representatives of United States industry, colleges, and government in discussing how advanced scientific feeding methods can increase production of animal products. The European specialists then left for the Midwest to visit land-grant institutions, federal and private laboratories, packing plants, and feed manufacturers.

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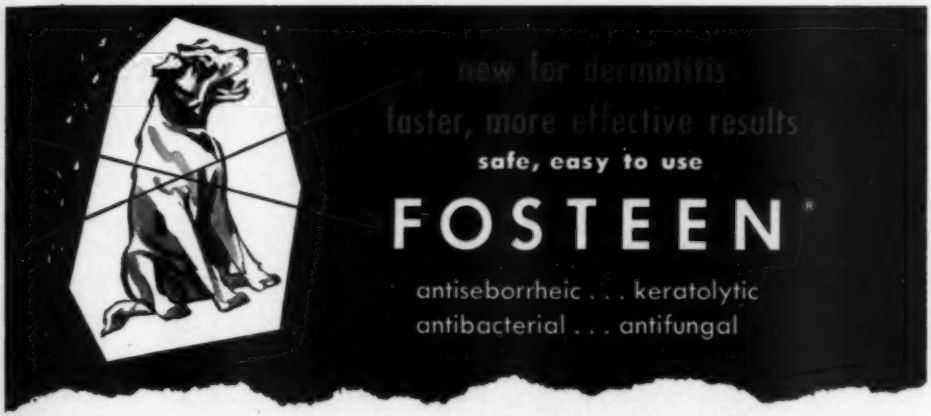

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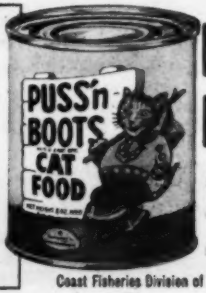

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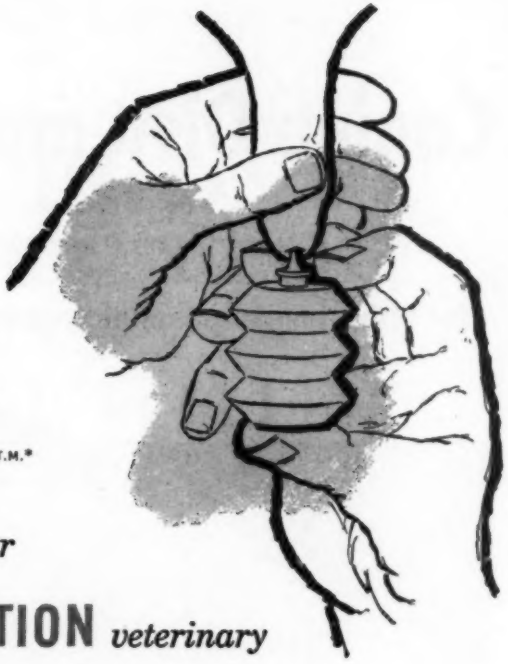

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1. Mires, M. H., and Chadwick, R. H.: Vet. News 10:3 (Jan.-Feb.) 1947. 2. Mires, M. H.: J. Am. Vet. M. Ass. 117:49 (July) 1950.

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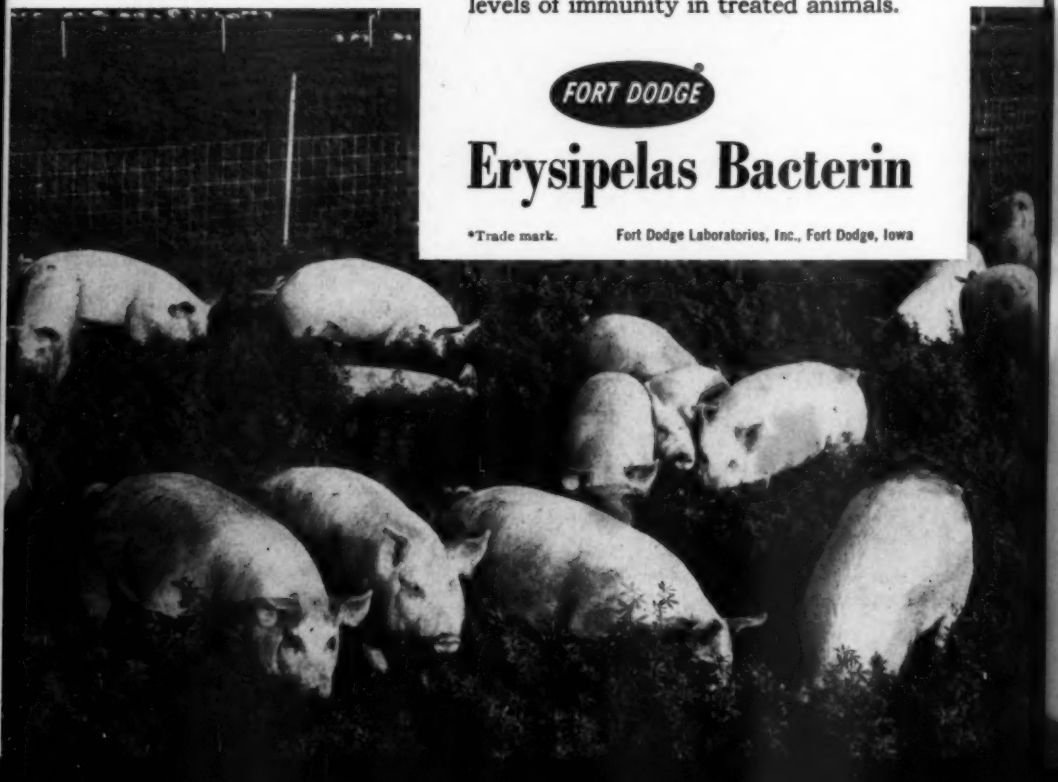

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Swine Repopulation. I. Performance Within a "Disease-Free" Experiment Station Herd

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STUDIES ORIGINATING in 1949 and reported early in 1951[15] demonstrated that baby pigs could be raised without colostral milk. Success in this venture was not based on any miraculous dietary formula but on a fundamental principle that had been neglected on previous attempts to raise pigs without colostrum. Lactoglobulins in colostrum are essential to survival of the baby pig in its natural environment because of the presence of a multiplicity of disease agents which can kill it. Lactoglobulins protect it against these diseases by *in vivo* neutralization of the disease-producing agents.

If the lactoglobulins are not provided, baby pigs must not be exposed to their dams' environment. This criterion was first met by catching pigs in sterile canvas bags at birth and removing them immediately to isolation rooms. Pasteurized cows' milk, whole chicken eggs, and minerals comprised a suitable diet for the early feeding of these pigs.

A series of elaborations in technique followed the discovery that baby pigs could survive without colostrum providing they were given a disease-free environment in which to begin life. Individual isolation units were devised to house "disease-free" and experimentally infected baby pigs.[16] A technique to obtain "disease-free" pigs by hysterectomy was developed,[19] and later an improved hood[11] for implementing this operation was created. A brooder[12] capable of housing 10 to 12 pigs in isolation until they were 4 weeks of age was also developed as part of the essential equipment for raising pigs in isolation.

It became obvious early in these studies that it was not difficult to raise "disease-free" pigs and that "disease-free" breeding stocks would be useful to replace stocks in herds plagued by chronic swine diseases. Although it is not feasible to repopulate the industry directly with pigs taken by hysterectomy, marked strides can be made by an indirect approach.[17] The essential steps in such a program are: (1) obtain aseptic pigs; (2) raise pigs in isolation; (3) grow pigs to maturity under farm isolation; (4) resume normal birth and production; and (5) restock other farms with "disease-free" pigs obtained by normal birth and production. All farms are managed to continuously isolate "disease-free" pigs from conventional pigs.

Application of these techniques to control certain diseases in the University of Nebraska herd was begun in the spring of 1956. Specific diseases present were atrophic rhinitis (AR) and virus pig pneumonia (VPP). One or more nonspecific intestinal ailments were also present. The techniques outlined have been successfully applied to the Nebraska Experiment Station herd. This paper is written to

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present the observations made and the results obtained in these studies.

MATERIALS AND METHODS

"Disease-Free."—A definition of "disease-free" swine is presented for clarity. The term is intended to indicate swine which have not been exposed to, or affected by, microorganisms capable of causing clinical illness. "Disease-free" is to be contrasted to "germ-free," which indicates freedom from all microorganisms. It is not possible to keep animals germ-free under practical husbandry conditions. Some bacteria are considered beneficial to the health of swine and no attempt is made to exclude them in "disease-free" herds.

Population.—The basic stocks used to repopulate the University of Nebraska Experiment Station herd were obtained from three pure breeds. These were Duroc, Hampshire, and Yorkshire breeds. They were obtained for the most part from the existing station herd. Sows were hand-mated to be certain of established breeding dates. This was an absolute must so that hysterectomies could be performed between 110 to 112 days after breeding. It is difficult to keep pigs alive when they are born less than 110 days after the sows are bred, and only a few pigs survive birth by hysterectomy when taken less than 108 days after conception.[18]

Management.—All pigs comprising the basic population in the "disease-free" herd were obtained by hysterectomy. The specific procedures of this technique have been described previously.[19] In general, this was the procedure used.

Sows 110 to 112 days past breeding were hoisted by their hindlegs. Their abdomens were washed thoroughly and rinsed well with water. The sows were then lowered head first into a barrel containing carbon dioxide gas. The gas was formed by crushing 3 lb. of dry ice in the bottom of the barrel and allowing the dry ice to disintegrate. Exposure to the gas was exactly one minute. The abdomen was washed with an antiseptic and cut open from the posterior teats to the zyphoid cartilage.

The gravid uterus was extended onto the external shelf of the hysterectomy hood,* freed from the dam by excision at the cervix, and passed into the hood through an antiseptic lock. Pigs were freed from their uterine encasement by grasping the uterus firmly with both hands (surgically clean) and tearing the uterus and fetal membranes away from each pig. This procedure was completed within 30 to 50 seconds after excision of the uterus from the donor dam. The pigs were wiped dry with sterile cloth sacks. Their navels were tied firmly with cotton cord to prevent excessive bleeding, cut about 1 inch long, and cauterized with tincture of iodine saturated with alum.

The pigs were placed in a carrying box and covered with heavy canvas while still within the hysterectomy hood. This carrying box, with enclosed pigs, was slid from the hood into a sterile canvas bag for transportation to the isolation quarters.

Newborn pigs obtained by hysterectomy were housed in individual isolation units for one week. Up to eight units were set up in a room on a single exhaust manifold.[16] The pigs were fed approximately ⅛ qt. (4 oz.) of a liquid diet from a shallow pan, morning, noon, and late afternoon. No night feedings were necessary. The diet consisted of 1 qt. of homogenized pasteurized cows' milk, yolk and white of one egg, and 5 ml. of a mineral mixture.** To this mixture was added 1 mg. of vitamin K (Klotogen F†) per quart of milk fed the first 24 hours. The milk mixture fed during this early period was also heated to 80 C., then allowed to cool so it could be fed without harm to the pigs. After 24 hours, the pigs consumed pasteurized milk with the low number of bacteria which it contained. A clean sterile pan was used for each feeding.

The pigs were placed in brooders[12] in groups of 10 to 12 at the end of one week. The liquid diet described was fed three times daily at a rate not to exceed ¾ qt. per pig per day. Prestarter pig pellets ("4 x 4" Automatic Pig Weaner‡) were present in a self-feeder within the brooder. Clean water was also continuously available. Most

*This hood and all equipment contacting pigs, i.e., isolation cages and brooders, were either sterilized with steam or formaldehyde gas following thorough physical cleansing. Warm air forced into the hood passed a filter capable of removing bacterial-sized particles.

**The mineral mixture consisted of 49.8 Gm. of $FeSO_4 \cdot 7H_2O$, 3.6 Gm. of $MnCl_2 \cdot 4H_2O$, 3.9 $CuSO_4 \cdot 5H_2O$, 0.26 Gm. of KI, and water to make 1 liter. Concentrated HCl is added in quantity to place in solution any precipitating salts.

†Klotogen F (stabilized menadione sodium bisulfite) is produced by Abbott Laboratories, North Chicago, Ill.

‡"4 x 4" Automatic Pig Weaner is made by Walnut Grove Products Co., Inc., Atlantic, Iowa.

of the pigs were eating prestarter in addition to milk by the time they were 2 weeks old.

One week in isolation units and three weeks in the brooder were adequate for sufficient adaptation of pigs for placement on the station farms. All pigs were vaccinated for hog cholera when 4 weeks old by use of rabbit-origin attenuated hog cholera virus and 20 ml. of anti-hog cholera serum. Serum does not interfere with developing immunity.[3] No other immunization procedures were used.

Pigs procured during the summer of 1956 were moved, when 4 weeks old, to a pasture which had been free of swine for the preceding five years. Pigs procured in the fall of 1956 were moved, when 4 weeks old, into a central growing-finishing unit which had been occupied six weeks earlier by the original contaminated herd.

The building and equipment were thoroughly cleaned and gassed with formaldehyde§ before re-use. Pig starter rations were fed until the pigs were 56 days old. Growing-finishing rations were then fed until the pigs were 154 days old. Formulas of the rations fed are shown (table 1).

Such pigs were used as the basic stock to resume ordinary swine raising practices. The performance of this primary hysterectomy stock was good and has been reported previously.[9] No contact was permitted between these pigs and pigs which had not originated by hysterectomy. Herdsmen wore only shoes and clothes which had not been worn while attending other swine. All visitors were provided with clean clinic boots and coveralls. Traffic to and from the premises was kept at a minimum.

Disease Evaluation.—Barrows marketed from the "disease-free" herd were examined at slaughter for presence or absence of lesions of AR and VPP. These observations were made because both AR and VPP were present in the herd before repopulation. Diagnoses were made as follows.

Pigs were individually tattooed at the station and slaughtered in a manner so that their identity was maintained. All snouts were sawed in cross-section and the nasal turbinates were examined carefully for evidence of atrophy. The lungs were examined grossly and tissue samples were taken from any abnormal or questionable areas. These tissues were later examined histopathologically for evidence of VPP.[13]

Fecal specimens from the pigs were examined by usual methods for eggs of internal parasites, and postmortem parasitological examinations were made on 5 pigs which succumbed to congenital defects or accidents. Livers from marketed pigs were observed for scars, which suggest previous migration of the larvae of *Ascaris suum*.

TABLE 1—Composition of Growing-Finishing Rations Fed to "Disease-Free" Swine

Ingredients	Amounts fed (lb.)	
	Up to 125 lb. weight	From 125 lb. to market weight
Ground yellow corn	1,581.0	1,712.0
44% solvent soybean oil meal	262.0	140.0
17% dehydrated alfalfa meal	50.0	50.0
50% meat and bone meal	50.0	50.0
Ground limestone	10.0	12.0
Steamed bonemeal	10.0	4.0
Salt (iodized)	10.0	10.0
Trace minerals (high zinc)	2.0	2.0
Hygromix	5.0	—
Vitamin premix*	20.0	20.0
	2,000.0	2,000.0
Calculated analysis:		
Crude protein (%)	14.0	16.0
Calcium (%)	0.73	0.73
Phosphorus (%)	0.53	0.61

*Contributed the following amounts of vitamins per pound of complete ration: vitamin D_3, 90 I.U.; riboflavin, 1.0 mg.; niacin 4.5 mg.; calcium pantothenate 2.0 mg.; choline chloride, 100 mg.; vitamin B_{12}, 5μg. In addition, 5 mg. of a tetracycline antibiotic was added per pound of feed to ration A.

RESULTS

Performance data from the station herd before and after repopulation with "disease-free" swine are presented (table 2). A better rate of gain at both 56 and 154 days was apparent in the performance of the "disease-free" stock. "Disease-free" pigs were approximately 30 per cent heavier at 56 days and 25 per cent heavier at 154 days than their predecessors in the same herd.

Pigs farrowed from dams that had been taken by hysterectomy were considered efficient in utilization of feed. Nutrition experiments (to be reported in detail elsewhere) show that 114 such pigs required only 3.09 lb. of feed per pound of gain from 35 to 135 days of age. Similar data from the "disease-free" breeding herd show that 47 Hampshire and 182 Yorkshire pigs required 2.90 and 2.94 lb. of feed per pound of gain, respectively, from weaning to market weight. The pigs on nutrition experi-

§Formula used was that prescribed for poultry incubators: 4 oz. of potassium permanganate in a small pail was covered with 1 pt. of formalin. One such unit was used for every 1,000 to 1,500 cu. ft.

ments were raised on concrete, whereas the pigs in the breeding herd were raised on pasture. This would account for part of the difference in feed efficiency between the two groups.

Fecal specimens from 33 pigs examined when they were 5 months old disclosed eggs of *A. suum* in 20 of the specimens. Two of the 5 pigs examined postmortem were infected with *A. suum*.

Examination of the noses, lungs, and livers from "disease-free" barrows at slaughter revealed that AR and VPP had not recurred in the repopulated stocks. Evidence of ascariasis was suggested by the presence of many scarred livers, especially in pigs marketed in the spring and fall of 1957. Management changes, which included use of a new anthelmintic,[6] resulted in virtual elimination of damaged livers in the nutrition herd for the spring of 1958. Striking characteristics of this herd were the complete lack of infestation with ectoparasites and the absence of other abnormal skin conditions.

TABLE 2—Comparison of Performance in Swine Before and After Repopulation

Year	No. of litters	Fertility and survival (%)		Weights (lb.)				Av. daily rate of gain
		Pigs born alive	Pigs 56 days	Av. wt./pig*		Av. litter wt.		56-154 days
				56-day wt.	154-day wt.	56-day wt.	154-day wt.	(lb.)
Before repopulation with "disease-free" stock								
Fall '55	80	11.4	8.3(73%)	29.8	183	247	1,521	1.57
Spring '56	63	11.3	9.1(80%)	31.8	171	289	1,554	1.42
After repopulation with "disease-free" stock								
Spring '57	34	9.1	7.7(85%)	44.9	199	346	1,535	1.58
Fall '57	29	10.4	7.1(68%)	36.8	223	261	1,581	1.90
Spring '58	23**	9.6	8.3(87%)	40.8	219	339	1,815	1.82
Spring '58	26†	10.2	8.9(86%)	41.3	215	367	1,913	1.77

*Birth weight averages ranged from 2.6 to 2.7 lb.; **nutrition herd; †breeding herd.

DISCUSSION

One apparent weakness in the evaluation of performance of "disease-free" pigs is inability to use direct controls. Ideally, the old diseased herd and the new "disease-free" herd should be evaluated at the same time on the same premises under identical management. This is not possible because of the transmissibility of disease. Thus, the establishment of a standard of performance, such as weight at given intervals, must serve as a criterion for evaluation.

Adaptation of "disease-free," colostrum-deprived pigs to farm-type management has been much easier than might have been expected. Many have expressed the opinion that laboratory-pampered pigs cannot survive in an environment as complex as the ordinary farm. This has proved to be an incorrect assumption since survival of pigs raised in the laboratory four weeks and then transferred to farms has been higher than that for naturally farrowed pigs. Several factors influence this high survival.

One factor which favors survival of the "disease-free" pig in a farm environment is an increased resistance to disease with an increase in age. The laboratory pig encounters a relatively simple environment. His first food is sterile, as is his environment. His first exposure to bacteria consists of those present in pasteurized milk which are considered harmless. Introduction of a prestarter ration with a somewhat more complex bacterial flora as the pig grows older aids him in his gradual environmental adaptation. This obvious adaptation to a more and more complex environment free of pathogenic bacteria and viruses is supplemented by natural age resistance plus an ability at 3 to 4 weeks to produce antibodies.

An example of this is the susceptibility of pigs to transmissible gastroenteritis (TGE) virus. Nearly all pigs less than 1 week old nursing nonimmune mothers die on exposure to TGE. Two-week-old pigs become very sick but survive. Four-week-old pigs, although infected as shown by indirect means,[14] may show no external evidence of disease.

To presume that "disease-free" pigs will encounter endless difficulties is not based on sound reasoning. "Disease-free" pigs, especially those that were farrowed naturally and which have nursed their mothers,* will be no more susceptible to a given disease than any other pig which

*This qualification is made because colostrum-deprived pigs do not produce antibodies as rapidly as pigs that have suckled.[4]

has not encountered that specific disease before. Thus, the "disease-free" herd is no more susceptible to hog cholera, erysipelas, or influenza than any ordinary herd. Actually, the "disease-free" herd is less vulnerable because the simultaneous occurrence of diseases tends to block the antibody-producing centers. Examples of this type are an increased susceptibility to the enterotoxins of organisms associated with infections with Brucella[1] and secondary bacterial infections complicating hog cholera immunization.[2]

It must be recognized that the live pig is the natural reservoir for most of our troublesome swine diseases. Viruses live only a short time outside the host cells, so that breaking direct contact with the diseased live pig does much to control diseases. Repopulation is based on prevention of contact with other pigs.

It is further based on providing a safe time interval between the presence of the diseased and the "disease-free" pigs on the same premises so that disease germs can be destroyed by natural exposure. An interval of six weeks during the summer months is considered adequate. During cool damp weather, eight to ten weeks is preferable.

It must be remembered that eggs of *A. suum* are highly resistant to drying and to chemical agents. These eggs can survive in the soil of hog lots for several years. Thus the methods presented in this paper, although they effectively eliminate some diseases, will not prevent infections with A. *suum* or parasites which have a long-lived intermediate host. For example, the earthworm is the intermediate host for Metastrongylus species or the swine lungworm. Lungworm larvae may persist in earthworms beyond four years.[8]

Breeding stocks free of chronic diseases such as AR and VPP give researchers in swine nutrition and genetics an increased opportunity for basic research through use of a superior experimental animal. Little research has been done on the nutritive requirements of swine without some degree of interference from disease. The possibility exists that our current nutritive levels are based on supplying nutritives for microorganisms in diseased swine as well as for growth of the pig. Evidence has already been presented that diets of "disease-free" pigs can be simplified without impairing growth of swine.[10]

The value to the geneticist of investigating the reactions of the "pure animal" have also been previously suggested.[7] Selection of stock for superior performance is often difficult because of adversities of the environment in which the pigs are raised. With the presence of a high disease level, the breeder is unable to discern whether the higher performing animals selected actually possess genetic resistance or if they have not been exposed, unless deliberate steps are taken to insure uniform infections. The latter is not a common practice in swine breeding research. A reduction in disease level may lead to some reduction in phenotypic variability and augment a more clear-cut determination of genetic difference in other aspects of performance. The importance of research in selection for disease resistance is not denied,[5] but is considered worthy of separate research projects by adequate experimental techniques.

SUMMARY

A "disease-free" swine herd was established by obtaining pigs by hysterectomy one to three days prematurely and raising them without colostrum, in isolation for four weeks. These pigs were placed in a clean farm environment, without direct or indirect contact with other swine, raised to maturity, mated, and the next generations were farrowed normally and permitted to nurse their mothers.

Naturally farrowed pigs from dams obtained by hysterectomy had high levels of performance. In 112 litters, during 1957 to 1958, 8 pigs per litter were raised with average weights of 41.0 lb. when weaned, at 56 days, and of 213 lb. at 154 days. The average daily gain was 1.75 lb. Feed efficiency was not determined for all pigs. The conversion ratio was essentially 3 lb. of feed for 1 lb. of gain as judged by tests on 379 pigs farrowed in the spring of 1958.

Atrophic rhinitis (AR) and virus pneumonia of pigs (VPP) were eliminated from the herd by the procedures followed. Examinations of lungs and nasal cavities for evidence of AR and VPP, in pigs going to slaughter, disclosed no lesions characteristic of these diseases.

The methods of procuring and raising "disease-free" pigs are simple enough to be used in a practical program to eliminate

or greatly reduce the incidence of AR and VPP in swine throughout the nation.

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Swine Disease Survey in Iowa

A poll of swine owners in Iowa indicated that erysipelas was the most prevalent swine disease during 1958. It was reported by 24 per cent, followed by scours in baby pigs (21%), influenza (18%), pneumonia (8%), enteritis of all kinds (5%), atrophic rhinitis (4%), hog cholera (3%), and jowl abscesses (1%). Absence of disease in their swine was reported by 27 per cent.

Of those reporting, 47 per cent had their pigs vaccinated for cholera with a modified virus and 22 per cent with live virus plus serum; 44 per cent had vaccinated for erysipelas, and 4 per cent for leptospirosis. —*Wallaces Farmer (Feb. 7, 1959): 11.*

The Top Swine-Raising Counties

Of the 100 counties in the United States which lead in the number and value of swine raised, 60 are in Iowa, 18 in Illinois, 6 each in Indiana and Minnesota, 4 in Wisconsin, 3 in Ohio, 2 in Nebraska, and 1 in Missouri. Henry County, Illinois, led with an estimated 318,000 in 1954, when the latest figures were compiled, followed by five Iowa counties each with more than 240,000 swine.—*Nat. Hog Farmer (April, 1959): 13.*

A Large "Disease-Free" Swine Herd

The first large commercial hog farm known to be using the disease-free, hysterectomy and sanitary brooder, method developed at the University of Nebraska (*J.A.V.M.A., 131,* (1957): 222, 279) is in operation in Jasper County, Missouri. The owner, a physician, is a "baby specialist."

The farm now has 250 sows bred so that about five litters are started each week. It is planned to increase to 450 litters per year.—*Farm J. (May, 1959): 59.*

More States Restrict the Use of Virulent Hog Cholera Virus

Five mainland states (Iowa, Oregon, South Dakota, Texas, and West Virginia) have been added to the list of 18 previously reported states (*J.A.V.M.A.*, Feb. 1, 1959: 145) which have restricted the use of virulent virus for vaccinating against hog cholera. Of the 23 states, less than a third have completely banned the importation and use of virulent virus. The degree of restriction

in the other affected states varies. More than two thirds of the nation's swine are raised in these 23 states.

In Iowa, where about 20 per cent of the nation's swine are raised, the new law forbids the sale or use of "virulent blood or virus from cholera-infected hogs. . ." except for out-of-state shipment, research, testing of biological products and for hyperimmunizing hogs. It can be used for vaccinating in special cases when, in the judgment of the authorities of the School Department of Veterinary Medicine, Iowa State College, an outbreak can not be controlled without simultaneous vaccination with the virulent vaccine.

Bills to restrict the use of virulent virus have been introduced in Colorado, Minnesota, Nebraska, North Carolina, and Pennsylvania.—*U.S.D.A.*

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Production of Virulent Hog Cholera Vaccine Discontinued by Jen-Sal.—Jensen-Salsbery Laboratories, Inc., of Kansas City, Mo., has discontinued production of virulent hog cholera virus. The chief reasons were that nearly half of the states have outlawed, or restricted, its use and it was used in only 18 per cent of the swine vaccinated in 1958. Instead, the company is marketing a modified live virus hog cholera vaccine, S.V.2, to be used with smaller doses of anti-hog cholera serum.—*Jen-Sal Release, Feb. 16, 1959.*

Simultaneous Vaccination Against Hog Cholera and Erysipelas

All of 34 susceptible swine were solidly immunized against hog cholera when simultaneously vaccinated with a formol-adsorbate cholera vaccine and an avirulent live erysipelas vaccine regardless of the season. However, the immunity to erysipelas varied in intensity, being complete in pigs vaccinated during the warm season but incomplete (54.5%) in those vaccinated from September to April. All of the similar swine vaccinated at the same cold season against erysipelas only were immunized.

Simultaneous vaccination against these two diseases is discouraged, and an interval of at least 14 days is suggested.—*M. Stanca et al. in Scientific Papers from Pasteur Serum and Vaccine Institute, Bucharest, 3, (1958): 229.*

Interference of Anti-Hog Cholera Serum with Immunity Induced with Erysipelas Bacterin

Immunity against swine erysipelas was less solid in areas of Yugoslavia where pigs were vaccinated simultaneously against erysipelas and cholera when anti-hog cholera serum was used.

Tests proved that ten of 11 commercial lots (91%) of anti-hog cholera serums examined contained antibodies against swine erysipelas. One lot (9%) had a titer of 4.65 immunity units and showed ability to lower the immunizing capacity of commercial adsorbed bacterin by 47 per cent.

Since anti-hog cholera serum, being homologous, is eliminated slowly, vaccination for erysipelas should either be postponed more than two weeks after its use, or a slowly adsorbed erysipelas vaccine should be used.—[*Z. Forsek, M. Zeljko, and Z. Romic: Interference of Anti-Hog Cholera Hyperimmune Serum with the Immunity in Mice Vaccinated Against Swine Erysipelas with Adsorbed Bacterin. Am. J. Vet. Res., 20, (May, 1959): 558-561.*]

A Bivalent Serum Against Hog Cholera and Erysipelas

Swine were hyperimmunized against erysipelas and hog cholera, thus producing a bivalent serum. The use of these antigens simultaneously did not interfere with formation of antibodies against both diseases.—*A. Nica et al. in Scientific Papers from Pasteur Serum and Vaccine Institute, Bucharest, 3, (1958): 127.*

The Tonsil and Swine Erysipelas

Erysipelothrix rhusiopathiae are probably destroyed in the lymph nodules of the tonsils of healthy pigs; however, in pigs weakened by stress, the organism can penetrate the vascular system of the tonsils and produce septicemia. Pigs that have died from erysipelas showed a necrosis of the tonsilar lymph nodes.—*Vet. Bull. (Feb., 1959): Item 316.*

Tetanus Immunity Transfer to Foals

Serological tests of a number of foals, whose dams had been previously immunized against tetanus, showed that a degree of immunity sufficient to protect the foals was present for a period of 32 days.—*Vet. Ital. (Jan., 1958): 16.*

A Serological Survey for Leptospiral Antibodies in Maryland Cattle

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THE FINDING of bovine leptospirosis in the United States has led to numerous serological surveys to determine the prevalence of infection. These surveys, most of which employed only *Leptospira pomona* as test antigen, indicated that bovine leptospiral infections were widespread.[2,3,7,12,16,17,25] The extent of bovine disease associated with leptospiral infections has been pointed up by the U.S.D.A. which estimated an annual financial loss of $112,282,000.[19]

Leptospira pomona has been implicated as a major cause of disease in cattle and swine in the United States, Australia, and New Zealand. Since *L. pomona* was the first serotype isolated from cattle in the United States and has been implicated more often than other strains in clinical bovine leptospirosis, it has been widely utilized as an antigen and vaccine in the detection and control of leptospiral infections. Swine and cattle are probably the principal animal reservoirs of *L. pomona*, although recent surveys have implicated a number of wildlife species as carriers.[8]

A number of other serotypes produce disease in cattle and there is evidence of their presence in North America, as demonstrated by isolations from animals or positive serological findings.

REVIEW OF THE LITERATURE

Leptospira grippotyphosa, the first serotype known to affect cattle, was seen in the Soviet Union in 1935 and was associated with a disease known as bovine icterohemoglobinuria. It has been found in many parts of the world and is the principal cause of bovine leptospiral infections in parts of Europe and the Middle East. In the United States, it has been recently isolated from the raccoon;[8] and elsewhere, from the rat, vole, and mouse. The evidence of *L. grippotyphosa* infections in cattle in the United States is based on positive findings in serological surveys.[7]

Leptospira canicola has been associated with leptospiral infections in dogs, and is transmissible from dogs to other animals. It has been found to be the cause of bovine leptospirosis in Israel[20] and produces a clinical disease comparable in severity to infections caused by *L. pomona* and *L. grippotyphosa*. In the United States, *L. canicola* has been isolated from a diseased calf, and also from man during an epidemic of leptospirosis due to *L. canicola*, which originated when infected cattle and swine had contaminated surface waters in which people swam.[22] The strain has also been isolated from the jackal[20] and rat.[21]

Leptospira icterohaemorrhagiae has been isolated from rodents and other mammals throughout the world.[6] It was the first leptospiral serotype to be isolated and is the causative agent of the classical Weil's disease in man. Both experimentally and in nature, it has caused clinical bovine leptospirosis.[4,6]

Leptospira autumnalis is distributed throughout the Far East and has been isolated from man and animals in the United States. It is associated with a disease syndrome in man variously known as autumnal fever, pre-tibial fever, or Fort Bragg fever. This serotype has been isolated from various species of Microtus in Japan and the Soviet Union, the bandicoot in Malaya, the raccoon and opossum in the United States,[8] and from cattle in Japan.[24]

Leptospira byos has been isolated in Australia, South America, and Europe. It has been chiefly associated with disease in man and swine,[14] although it has been isolated from cattle in Australia[23] and there is serological evidence of its presence in cattle in Argentina.[13] There has been a recent isolation of a closely related strain known as *Leptospira bakeri* from the opossum in the southern United States.[9]

Leptospira sejroe, a member of the hebdomadis serogroup, has been isolated from man and one species of rodent in northern Europe. Antibodies to *L. sejroe* have been detected in cattle in Europe, Canada, and the United States. Paradoxically, the only serotype thus far isolated from cattle with antibodies to *L. sejroe* has been *L. pomona*, a species antigenically unrelated to *L. sejroe*.[1] The pathogenicity of *L. sejroe* for cattle was indicated when disease and abortion followed injection with this serotype.[5,12] The inoculated cattle developed specific antibodies to *L. sejroe* and did not develop antibodies to *L. pomona*.

A serological survey was conducted for leptospiral antibodies in Maryland cattle in the summer of 1957. Antigens were selected from serotypes previously associated either with disease in cattle or positive serological findings.

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MATERIALS AND METHODS

The serum specimens tested in this survey were obtained from samples previously submitted to one of the Maryland livestock diagnostic laboratories as part of the state-wide brucellosis eradication program. Specimens were obtained from 1,998 cattle in 128 herds from 15 of Maryland's 23 counties, without reference to herd histories or suggestion of previous disease. Most of the sampling represented entire herds, although there were some specimens included from smaller groups or individual animals requiring health certificates.

Selection of specimens was at random within counties, but certain counties of the state were sampled more than others because of their proximity to the laboratories. The serums which were not tested on the day they were received were frozen and stored at −40 C.

The agglutination-lysis test was conducted essentially as previously described.[10] The antigens employed were *L. pomona* (V42), *L. hyos*, *L. sejroe* (Mallersdorf), *L. icterohaemorrhagiae* (Wijnberg), *L. grippotyphosa* (Moscow V), *L. canicola* (Ruebush), and *L. autumnalis* (FBF).* The entire complement of seven antigens was employed routinely in the testing procedure.

*Original cultures were furnished by the Division of Veterinary Medicine, Walter Reed Army Institute of Research, Walter Reed Army Medical Center, Washington, D.C.

The serum samples were initially diluted 1:50 with buffered saline solution, and to each 0.2 ml. was added 0.2 ml. of antigen, giving a final serum dilution of 1:100. The serum-antigen mixtures were allowed to stand at room temperature for two hours and then read by placing a drop on a glass slide and examining it by dark-field microscopy at a magnification of 150 x.

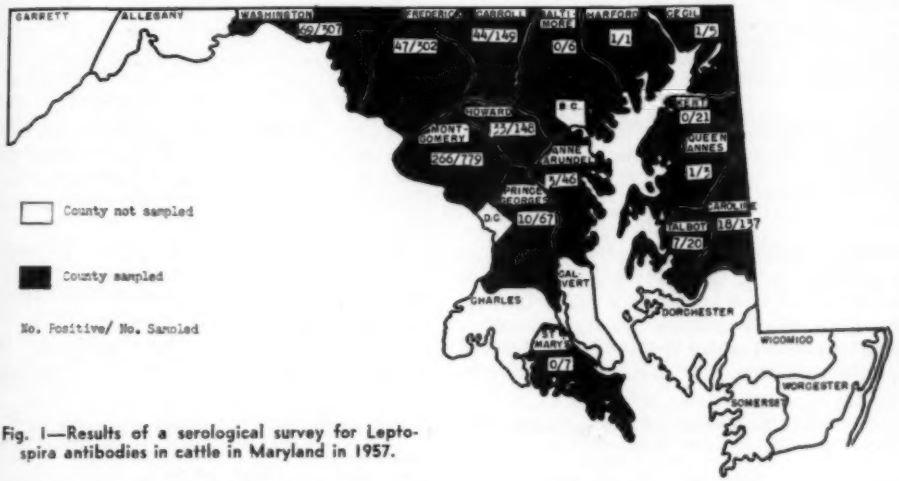

Fig. 1—Results of a serological survey for Leptospira antibodies in cattle in Maryland in 1957.

The reaction was considered positive when at least 50 per cent of the organisms were agglutinated at the lowest serum dilution employed. Those serum specimens which were positive at the screening dilution of 1:100 were serially diluted in fourfold increments so that end points were reached. The highest dilution at which they were positive was considered the titer and is recorded in the results. The antigens were periodically tested against known positive antiserums which

TABLE 1—Distribution of Cattle Serologically Positive for Leptospirosis, According to Size of Herds Tested

Animals per herd	No. of herds	No. of animals	Positive herds (No.)	Positive herds (%)	Positive animals (%)	Positive animals (No.)
< 10	68	263	29	43	66	25
11-20	26	388	24	92	86	22
21-30	14	324	11	79	76	23
31-40	7	247	7	100	83	34
41-50	7	324	7	100	98	30
> 50	6	452	6	100	91	20
Totals	128	1,998	84		500	

TABLE 2—Comparison of Single and Multiple Agglutination Reactions in 500 Serologically Positive Specimens from Cattle Tested for Leptospirosis

Antigen	Multiple*			Total multiple reactions	Single reactions**
	Dual	Triple	Quadruple		
L. pomona	73	13	2	88	155
L. sejroe	46	6	1	53	211
L. grippotyphosa	8	6	1	15	13
L. hyos	3	2	0	5	3
L. canicola	1	0	0	1	6
L. icterohaemorrhagiae	4	2	2	8	2
L. autumnalis	33	13	2	48	10
Total multiple reactions	168	42	8	218	400
Total specimens	84	14	2	100	500

*Specimens reacting with indicated antigen and one, two, or three others; **specimens reacting only with indicated antigen.

were prepared as described.[1] Antigen controls were included with each day's testing.

RESULTS

The prevalence of leptospiral antibodies is shown (fig. 1, table 1), and data on the antigens involved in positive agglutination reactions and the serum titers encountered are presented (tables 2, 3, and 4). Of 1,998 specimens tested, 500 (25%) were positive against one or more leptospiral serotypes, at least at the lowest serum dilution employed. Of 128 herds or groups, 84 (66%) contained serologically positive animals. The use of seven test antigens doubled the number of reactors that would have been detected had only *L. pomona* antigen been used.

The total *L. sejroe* reactions exceeded those involving *L. pomona* (table 2). However, *L. pomona* was encountered more frequently in multiple reactions than *L. sejroe* (tables 2, 3). Four antigens (*L. grippotyphosa, L. hyos, L. icterohaemorrhagiae,* and *L. autumnalis*) co-reacted with other serotypes more often than they appeared as single reacting antigens (table 2).

TABLE 3—Analysis of Dual Reactions in 84 Serum Samples from Cattle Tested for Leptospirosis

Antigen A	Antigen B	No. with equal serum titer	No. with higher titer to:		Totals
			(antigen A)	(antigen B)	
L. pomona	L. sejroe	20	13	3	36
L. pomona	L. autumnalis	17	14	1	32
L. pomona	L. grippotyphosa	2	1	0	3
L. pomona	L. icterohaemorrhagiae	2	1	0	3
L. sejroe	L. grippotyphosa	2	3	0	5
L. sejroe	L. hyos	1	2	0	3
L. autumnalis	L. sejroe	0	0	1	1
L. canicola	L. icterohaemorrhagiae	1	0	0	1
					84

TABLE 4—Antibody Titers of 500 Serologically Positive Specimens* from Cattle Tested for Leptospirosis

Antigen	Dilution of serum				Totals
	1:100	1:400	1:1,600	1:6,400	
L. sejroe	170	87	5	0	262
L. pomona	133	91	17	4	245
L. autumnalis	47	9	1	1	58
L. grippotyphosa	22	6	0	0	28
L. icterohaemorrhagiae	8	1	1	0	10
L. hyos	7	1	0	0	8
L. canicola	5	1	0	1	7
Totals	392	196	24	6	618**

*Includes serums reacting to only one antigen (400) as well as those reacting to two or more (100); **includes single reactions (400) and multiple reactions (218).

In those serum specimens reacting with two leptospiral serotypes, *L. pomona* not only appeared more frequently than other strains but tended to predominate in regard to serum titers (table 3). The strains appearing most frequently with *L. pomona* in dual reactions were *L. sejroe* and *L. autumnalis*.

Serum titers at least as high as 1:400 were observed for all seven antigens used (table 4). Three serotypes (*L. pomona, L. autumnalis,* and *L. canicola*) reacted with specimens at titers as high as 1:6,400.

DISCUSSION

Since the reported incidence of clinical bovine leptospirosis in Maryland is less than might be expected from results of this serological survey, it is likely that inapparent or subclinical leptospiral infections are frequent, particularly in adult cattle. The effect of leptospirosis vaccination on these results has been considered, although other reports indicate that high, persistent agglutination titers are not elicited from the vaccines currently in use. Cattle in Maryland are usually vaccinated for leptospirosis only in the event of an outbreak of clinical disease and not as a routine procedure.

Although *L. pomona* infections have been commonly associated with clinical bovine leptospirosis in the United States, the significance of reactions to other serotypes is still undetermined and warrants further study. The disease-producing capacity of *L. grippotyphosa, L. canicola,* and *L. icterohaemorrhagiae* for cattle has been clearly established. Although *L. sejroe* has never been isolated from naturally infected cattle, this species is a member of the hebdomadis serogroup of which the type strain *L. hebdomadis* has been incriminated in clinical bovine leptospirosis.

The high number of reactions to *L. autumnalis* might be explained on the basis of the antigenic relationship of that serotype to *L. pomona.* Conceivably, many *L. autumnalis* titers are the result of past *L. pomona* infection in the animal tested. Analogous observations have been made in other animal species.[11]

The reported isolation of *L. pomona* from a cow serologically positive to *L. sejroe*[7] suggests the possibility of paraspecific reactions in animals infected with *L. pomona.* Aside from these cases, it is still reasonable to expect that the majority of positive serological reactions represent past infection of animals with the reacting serotype or an antigenically related organism.

SUMMARY

Cattle in 15 of Maryland's 23 counties were tested for antibodies using seven leptospiral serotypes as antigens. Of 1,998 serum specimens tested in 128 herds, 500 (25%) from 84 herds (66%) were positive at 1:100 dilution or higher against one or more leptospiral antigens employed. If *Leptospira pomona* alone had been used as the test antigen, only one half of these reactors would have been detected.

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Respiratory Diseases in Feedlot Cattle

Diseases of the respiratory system commonly affect feedlot cattle. Pneumonia is almost always present but is not serious except during the hot, dry months when the lots become dusty. Dust pneumonias are difficult to treat. In addition to sulfonamides and antibiotics, atropine sulfate and antihistamines have great value because they dilate the bronchioles and diminish the copious secretions of mucus.

Shipping fever is quite common in calves, but does not severely affect yearlings and older cattle. No biological agents are used specifically for the control of shipping fever. However, since we have been vaccinating all incoming cattle for infectious bovine rhinotracheitis, the incidence of shipping fever has decreased greatly.

In calves, other measures for reducing the incidence of shipping fever are used: 1.5 million units of penicillin is given to calves soon after arrival; tranquilizers given to those coming into the lots helps in getting them adapted to the feedlot and on feed more quickly.

Calf diphtheria is common and occurs at any time during the feeding period. Either broad-spectrum antibiotics or sulfonamides are given intravenously. Additionally, a mixture of proteolytic enzyme, penicillin, and streptomycin is injected into the lumen of the trachea as near the larynx as possible.

Infectious bovine rhinotracheitis was the most serious disease encountered in the feedlot before the vaccine was available. Now all cattle coming into the lots are effectively vaccinated against this disease. Treatment would be similar to that for calf diphtheria.

Rhinotracheitis and diphtheria may be difficult to differentiate. Rhinotracheitis is characterized by a morbidity of 15 to 20 per cent, body temperature from 106 to 108 F., and lesions extending from the nasal cavity to and including the trachea and varying from severe hyperemia to necrosis of the mucous membrane. In diphtheria, the morbidity is about 2 to 5 per cent, temperature 104 to 106 F., and the lesions consist of abscesses and necrosis of the vocal cords and the arytenoid cartilages. These lesions can easily be seen by means of a Frick speculum and a pen light.—*E. H. Scheele, D.V.M., Greeley, Colo., at the Feb., 1959, meeting of Colorado V.M.A.*

Virus Pneumonia in Cattle

Pneumonitis, also known as "cuffing pneumonia" or virus pneumonia, was reported in 92 cattle from 85 different herds in Denmark. The majority were calves 1 to 12 months old. Most of the herds reported calf mortality or respiratory ailments in adult cattle. Small areas of pulmonary consolidation and atelectasis were characteristic. Histologically, there were peribronchial and perivascular accumulations of lymphoid cells, histiocytic cells in the bronchial mucosa, and alveolar septa, proliferation of alveolar cells, and catarrhal bronchitis.

The intestinal mucosa was frequently thickened and sections of small intestines showed cellular infiltrations of the mucosa. Hyperplasia of the lymphoid tissue in the lymph nodes, Peyer's patches, and in the spleen was rather constant. The pneumonitis was benign in animals kept under favorable conditions.—*H. E. Ottosen in Nord. Vet.-med., 9, (1957): 569.*

A Random Serological Survey for Evidence of *Leptospira Pomona* Infection in Indiana Cattle

R. K. JONES, D.V.M., Ph.D.

Lafayette, Indiana

LEPTOSPIRA POMONA infections have been detected by serological or cultural examinations in swine, cattle, and horses in Indiana. During 1956 and 1957, veterinarians requested serological tests for 12,033 cattle in 1,516 herds. Compilation of the test results revealed that 10.1 per cent of the cattle had significant serological titers, and that 1 or more animals in 32.3 per cent of the herds had significant titers. Only a small portion of these tests was made of complete herds and, therefore, represented a biased sample (clinical signs suggestive of leptospirosis prompted the request for testing in an undetermined number of cases). It was felt that the figures failed to give accurate information regarding the actual over-all incidence of the disease, and the incidence within individual herds.

Serological surveys for *L. pomona* infection have been made in Ohio[1] and Illinois.[2] Results indicate that the disease is rather widely disseminated among the cattle and swine of these states. It has been indicated that swine are probably the reservoir of infection and that cattle have been infected by placing them in contact with experimentally infected swine.[3]

Livestock production, particularly of cattle and swine, is intensive in Indiana; however, the concentrations of livestock types vary in different areas of the state. The present study was made to determine possible correlations of the incidence of *L. pomona* infections in serological samples of cattle selected at random with the nature of the livestock enterprise, relative concentration of swine, cattle herd type and size, and geographic location within the state.

MATERIALS AND METHODS

Testing Procedure.—The plate agglutination test[4] was used as the means of detection of infection. Herds were considered infected when 1 or more animals within the herd were found to have titers of 1:160 or above. In such herds, animals with titers of 1:40 were also considered infected. Herds in which no animals were found to have titers as high as 1:160 were considered uninfected regardless of whether lower titers were detected.

Serum samples were screened by a pooling procedure; 0.2 ml. of serum from five or less samples of a given herd were combined and 0.01 ml. of the pool was mixed with 0.03 ml. of antigen* and the reaction recorded. This method expedited the testing of serums in that it would detect the presence of a titer of 1:40 or more in any one of the five samples pooled. In the event that a titer was detected by this method, the samples making up the pool were tested individually.

If titer was detected at screening, the serum was diluted 5, 20, 40, and 80 times with saline solution,

Fig. 1—Counties from which samples of cattle serums were selected at random: Arabic numerals—county number; D—predominating dairy enterprise; B—predominating beef enterprise; S—predominating swine enterprise; M—mixed livestock enterprise; Roman numerals—geographic classification.

From the Department of Veterinary Science, Agricultural Experiment Station, Purdue University, Lafayette, Ind. Journal paper No. 1380.

*Leptospira pomona antigen, Fort Dodge Laboratories, Inc., Fort Dodge, Iowa.

TABLE 1—Incidence of Positive Serological Tests for Leptospira Pomona in Cattle in 27 Counties in Indiana

County No.	Milk cows* (thousands)	Approximate animal ratio;* dairy: beef: swine	Livestock classification**	Geographic classification**	Cattle tested (No.)	Herds tested (No.)	Cattle positive (No.)	Herds positive (No.)	Cattle positive (%)	Herds positive (%)
1	6.8	1.0:1.0: 1.5	Dairy	V	408	19	0	0	0.0	0.0
2	7.9	1.0:1.0: 2.0	Dairy	V	618	37	0	0	0.0	0.0
3	12.4	1.0:1.0: 3.5	Dairy	V	354	18	0	0	0.0	0.0
4	2.5	1.0:6.0:15.0	Beef	IV	966	105	6	4	0.6	3.8
5	4.5	1.0:5.0:11.0	Beef	IV	1,038	76	23	5	2.2	6.6
6	9.7	1.0:1.5: 6.0	Dairy	V	916	54	14	3	1.5	5.5
7	12.3	1.0:1.8: 6.5	Dairy	V	1,188	86	2	2	0.2	2.3
8	2.3	1.0:8.0:23.0	Beef	IV	1,901	88	10	2	0.3	2.2
9	4.9	1.0:5.0:24.0	Swine	III	315	23	0	0	0.0	0.0
10	2.3	1.0:5.5:16.0	Beef	IV	1,507	70	7	6	0.5	8.6
11	3.8	1.0:7.0:30.0	Swine	III	1,477	109	11	7	0.7	6.4
12	5.2	1.0:3.0:24.0	Swine	III	628	38	8	4	1.2	10.5
13	4.7	1.0:4.0:22.0	Swine	III	1,008	70	4	3	0.4	4.2
14	8.8	1.0:2.0:11.0	Mixed	III	1,074	65	11	5	1.0	7.7
15	11.3	1.0:0.9: 3.0	Dairy	III	1,058	95	3	2	0.2	2.1
16	1.8	1.0:9.0:10.0	Beef	IV	467	29	19	6	4.0	20.6
17	3.2	1.0:3.0:14.0	Mixed	III	1,722	116	14	7	0.8	4.3
18	3.8	1.0:3.0:25.0	Swine	III	627	43	5	3	0.8	6.9
19	9.5	1.0:1.7:10.0	Dairy	II	1,634	85	59	14	3.6	16.4
20	4.3	1.0:2.0:10.0	Mixed	III	1,596	96	26	7	1.6	7.2
21	6.3	1.0:3.8:26.0	Swine	II	1,106	75	29	13	2.6	17.3
22	3.0	1.0:3.0:17.0	Mixed	II	580	39	23	11	4.0	28.2
23	2.8	1.0:2.5:30.0	Swine	II	446	29	6	2	1.3	6.8
24	6.2	1.0:1.5: 4.8	Mixed	I	1,318	85	46	16	3.5	18.8
25	7.3	1.0:1.0: 2.0	Dairy	I	269	19	0	0	0.0	0.0
26	7.3	1.0:0.5: 0.5	Dairy	I	466	37	0	0	0.0	0.0
27	4.7	1.0:4.0:14.0	Beef	III	976	59	6	2	0.6	3.4
Totals					25,663	1,665	332	124	1.3	7.4

*Figures derived from 1954 U.S. Census of Agriculture; **see figure 1.

and 0.03 ml. of the diluted serum was mixed with an equal quantity of the antigen on a glass plate and the reaction was recorded after a six-minute incubation period. The occurrence of clumping (or the disappearance of homogeneous opalescence) in any degree was recorded as positive.

Serum Samples.—The serum samples were made available through the Brucellosis Testing Service.** The protocol of the survey required that each lot tested for evidence of *L. pomona* infection be a complete sampling of all breeding animals in the herd. The lots were then selected on the basis of county of origin (fig. 1), nature of herd (dairy, beef, or mixed on basis of predominating breed), and herd size (classified "large" if comprised of 10 or more animals, "small" if comprised of 9 or less animals).

Samples were selected from 27 counties which were considered representative areas in which a type of livestock enterprise (dairy, beef, swine, mixed swine, and beef) predominated. Such classification was determined on the basis of relative numbers of "milk cows," "steers and beef heifers," and "hogs and pigs" in the county, indicated by the 1954 agricultural census. An arbitrary geographic classification of the origin of samples was also made. These numbers and classifications are indicated (table 1).

Serum samples were collected over an 18-month period beginning in November, 1956.

TABLE 2—Incidence of Positive Tests for Leptospira Pomona in Cattle in Relation to Sampling Method

Samples tested	Cattle tested (No.)	Herds tested (No.)	Cattle positive (No.)	Herds positive (No.)	Cattle positive (%)	Herds positive (%)
Requested	12,033*	1,516	1,231	501	10.1	32.3
Random	25,663	1,665	332	124	1.3	7.4

*Total cattle samples submitted for routine testing Jan. 1, 1956-Dec. 31, 1957.

RESULTS

A total of 25,663 serum samples from 1,665 farms was tested, and 332 individual animals were found serologically positive. This was an over-all herd incidence of 7.4 per cent and an animal incidence of 1.3 per cent (table 1). No serological evidence of infection was found in six of the 27 coun-

**The cooperation of Dr. L. G. Morehouse, ADEB, ARS, U.S.D.A., is acknowledged.

TABLE 3—Incidence of Positive Tests for Leptospira Pomona in Cattle in Relation to Predominant Livestock Enterprise in County Groups

Predominating enterprise	Counties* included	Approximate animal ratio;** dairy: beef: swine	Cattle tested (No.)	Herds tested (No.)	Cattle positive (No.)	Herds positive (No.)	Cattle positive (%)	Herds positive (%)
Dairy	1,2,3,6,7,15,19,25,26	1.0:1.0: 2.6	5,723	364	76	19	1.3	5.2
Swine	9,11,12,13,18,21,23	1.0:4.0:25.9	5,607	387	63	32	1.1	8.2
Beef	4,5,8,10,16,27	1.0:6.9:14.7	7,855	427	71	25	0.9	5.9
Mixed	14,17,20,22,24	1.0:2.8: 8.9	7,468	487	122	48	1.6	9.9

*See figure 1 and table 1; **from 1954 U.S. Census of Agriculture (see table 1).

ties, although infection had been detected in five of them in either swine, cattle, or both in "requested" tests.

The difference in incidence of infection in "request" samples as contrasted to the randomly selected samples was highly significant (table 2).

There were no significant differences in incidence as related to predominating type of livestock enterprise (table 3).

There were significant differences in incidence as related to geographic location. The highest rate was in area II, a group of four east-central Indiana counties, an area in which swine raising predominates. However, the incidences in areas III and IV, in which swine production is also quite prominent, were considerably lower (table 4).

The incidence of herd infection was significantly greater in large herds, but this correlation was not reflected in the individual animal incidence (table 5).

There were no significant differences in the incidence as correlated to breed-type of herd (table 6).

DISCUSSION AND CONCLUSIONS

The over-all incidence of *L. pomona* infection in Indiana cattle appears to be somewhat lower than that found in serological surveys made in the adjoining states of Ohio[1] and Illinois[2] (table 7). However, since a presumably less sensitive method of testing was used in the survey reported here, direct comparisons probably should not be made. Since there is relatively good correlation between serological test results and clinical signs (table 2), we feel that the conclusions drawn in the remainder of the comparisons reported are valid.

Since the incidence of infection in cattle was not significantly higher in all areas having heavy swine population, swine may not be as important a source of infection for cattle as suggested by other workers. Information regarding the degree of direct association of cattle with swine in both infected and noninfected herds would be of value in drawing definite conclusions, but this information was not available.

TABLE 4—Incidence of Positive Tests for Leptospira Pomona in Cattle in Relation to Geographic Location

Area*	Counties* included	Approximate animal ratio; dairy:beef:swine	Cattle tested (No.)	Herds tested (No.)	Cattle positive (No.)	Herds positive (No.)	Cattle positive (%)	Herds positive (%)
I	24,25,26	1.0:1.0: 2.4	2,053	141	46	16	2.2	11.3
II	19,21,22,23	1.0:1.0:20.7	3,766	228	117	40	3.1	17.6
III	9,11,12,13,14, 15,17,18,20,27	1.0:3.4:17.7	10,481	714	88	40	0.8	4.9
IV	4,5,8,10,16	1.0:7.5:15.0	5,879	368	65	23	1.1	6.2
V	1,2,3,6,7	1.0:1.3: 3.9	3,484	214	16	5	0.5	2.3

*See figure 1 and table 1.

TABLE 5—Incidence of Positive Tests for Leptospira Pomona in Relation to Herd Size

Herd size	Cattle tested (No.)	Herds tested (No.)	Cattle positive (No.)	Herds positive (No.)	Cattle positive (%)	Herds positive (%)
1-9	3,204	626	30	18	0.9	2.9
10 & over	22,459	1,039	302	106	1.3	10.2

TABLE 6—Incidence of Positive Tests for Leptospira Pomona in Cattle in Relation to Breed-Type of Herd

Herd type	Cattle tested (No.)	Herds tested (No.)	Cattle positive (No.)	Herds positive (No.)	Cattle positive (%)	Herds positive (%)
Dairy	12,765	876	170	56	1.3	6.4
Beef	6,403	403	84	40	1.3	10.0
Mixed	6,495	386	78	28	1.2	5.2

Of the factors investigated, only geographic location appeared to be significant.

The relatively higher incidence of infection in large herds as contrasted to small herds can be explained by the probable greater movement of cattle into these herds. The highest incidence was not in large dairy herds in which the movement of cattle might be expected to be greatest.

Perhaps the most significant finding in the study is that of relatively low incidence in randomly selected herd samples as compared to the biased "request" sampling (table 2). Results indicate that the clinical signs of leptospirosis are recognized by practitioners and that the method of testing used is valuable.

Additional information is being collected regarding persistence of titers and spread of infection within herds. The preliminary evidence indicates that in most instances infection does not usually spread widely within herds.

TABLE 7—Incidence of Positive Tests for Leptospira Pomona in Cattle in Ohio, Illinois, and Indiana

State	Cattle tested (No.)	Herds tested (No.)	Cattle positive (No.)	Herds positive (No.)	Cattle positive (%)	Herds positive (%)
Ohio*	10,000	725	412	194	4.1	26.7
Illinois*	8,012	788	377	156	4.7	19.8
Indiana**	25,663	1,665	332	124	1.3	7.4

*Agglutination lysis test;[1,2] **plate agglutination test.

SUMMARY

1) *Leptospira pomona* infections in cattle as indicated by positive finding in plate agglutination tests of randomly selected serum samples were detected in 332 of 25,663 cattle tested in 1,665 herds. The over-all incidence of infection was 1.3 per cent on the individual animal basis and 7.4 per cent on the herd basis. These incidences are somewhat lower than those reported in adjacent states.

2) Attempts to correlate incidence of infection in individual cattle with high concentrations of swine, breed of cattle, and herd size were not positive, although the incidence of herd infection was greater when large herds were sampled.

3) There appeared to be a correlation of incidence of infection with geographic location within the state.

4) The results of random serological testing for evidence of *L. pomona* infections lend support to the acceptance of the use of the plate agglutination method of testing as a method of confirmation of diagnosis when clinical signs of the disease are observed in cattle.

References

[1]Bohl, E. H.: The Incidence and Clinical Aspects of Leptospirosis in Cattle and Swine in Ohio. Proc. Book, AVMA (1955): 167-169.

[2]Bryan, H. S.: Studies on Leptospirosis in Domestic Animals III. Incidence of Leptospirosis in Cattle and Swine in Illinois. J.A.V.M.A., *124,* (June, 1954): 423-426.

[3]Burnstein L., and Baker, J. A.: Leptospirosis in Swine Caused by *Leptospira Pomona.* J. Infect. Dis., *94,* (1954): 53-64.

[4]Stoenner, H. G.: Application of the Capillary Tube Test and a Newly Developed Plate Test to the Serodiagnosis of Bovine Leptospirosis. Am. J. Vet. Res., *14,* (1954): 434-439.

A New Breed Butterfat Record

A 9-year-old purebred Holstein-Friesian cow, in Michigan, recently completed a new breed record of 1,529 lb. of butterfat (33,718 lb. milk) in three times-per-day milking for 365 days. The average was 4 lb. of butterfat and 92 lb. of milk (11 gal.) per day.—*Hoard's Dairyman (April 10, 1959): 363.*

What Is Your Diagnosis?

Because of the interest in veterinary radiology, a case history and radiographs depicting a diagnostic problem are usually published in each issue.

Make your diagnosis from the picture below—then turn the page ▸

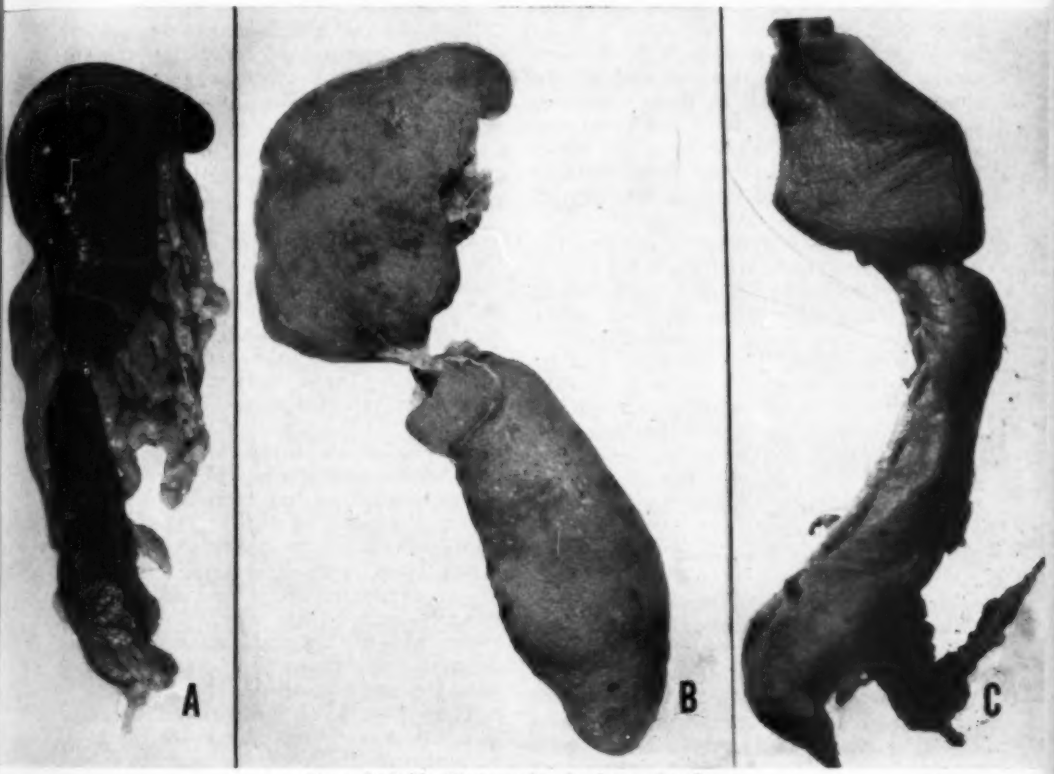


Fig. 1 (A,B,C)—Photographs of spleens of 3 dogs.

History.—During necropsies of dogs, occasionally a spleen is found that has been divided into segments (fig. 1 A, B, C). Other lesions or histories of previous traumatic accidents which could account for these defects are seldom if ever presented.

Here Is the Diagnosis

(*Continued from preceding page*)

Diagnosis.—These are thought to be examples of "developmental amputations," occurring as the result of locally restricted circulation. These are not examples of multiple or accessory spleens.

Comment.*—During the development of soft organs, such as the spleen, any pressure which is constant, even for a relatively short period, may produce developmental amputation as seen in each of the spleens shown (fig. 1). In these instances, strands of connective tissue and blood vessels which hold the segments of the organ together indicate that the defect-causing factor occurred after the morphology of the spleen had been established.

Multiple spleens are fairly common. In such anomalies, ordinarily the blood supply of each segment is distinct; the splitting of the anlage undoubtedly occurred before the rudimentary organ was established.

"It is recognized that the body and its organs always display some individuality in size, form, architecture, or position. When, however, an organ or organism clearly oversteps what can reasonably be accepted as a permissible range of variation, then the condition is known as an anomaly or malformation."[1]

The incidence of anomalies in animals is not known, but in man one in each 165 babies carries some major malformation, either external or internal.

This report was submitted by the Riser Animal Hospital, Skokie, Ill.

Our readers are invited to submit histories, radiographs, and diagnoses of interesting cases which are suitable for publication.

*Dr Harry L. Foust, professor emeritus and formerly head of the Department of Veterinary Anatomy, Iowa State College, Ames.

[1]Arey, L. B.: Developmental Anatomy. 5th ed. W. B. Saunders Co., Philadelphia, Pa. (1946): 172-173.

Irradiation of Transmissible Venereal Tumor of the Dog

In 8 female dogs, tumors in the vaginal wall adjacent to the labia disappeared completely following x-ray therapy given in six exposures of 200, 300, or 400 r over a period of three weeks (1,200 to 2,400 r total dose). From the results obtained, it was concluded that these tumors are sensitive to irradiation.

Histological studies revealed transient suppression of mitosis, cellular and nuclear enlargement, and mitotic aberrations in the earlier stages of treatment. Observed in the advanced stages of tumor regression were nuclear disintegration and infiltration with granulocytes, lymphoid cells, histiocytes, and plasma cells, followed by proliferation of fibrous tissue.

Both clinical and hematological observations indicated that irradiation therapy was well tolerated.—*Masaaki Hataya et al. in Jap. J. Cancer Res., 49, (Dec., 1958): 307.*

Rabies Increasing Along U.S.-Mexican Border

The United States leads the Americas in the recorded number of animal rabies cases with 2,706 reported for 1958, an increase of 20 over 1957. The states of Texas, New Mexico, Arizona, and California accounted for slightly more than one third of the total, with Texas having the greatest number of reported cases of rabies in the nation.

Several factors contribute to the seriousness of the border problem. The region's canine population has more than doubled and the number of border crossings has increased during the past few years. Another threat is the increasing appearance of a paralytic rabies carried by the vampire and other bats. Though long recognized in Latin America, the rabid bat was unknown north of the Rio Grande until five years ago.

These bats have great range and little is known about them. They are the cause of numerous livestock deaths from rabies, especially among cattle. The first death in man in the United States definitely attributed to bat rabies was reported in California last year.

A further complication centers around the skunk, which threatens to become the main source of both animal and human rabies because its natural enemies, the coyote and wolf (previously the principal wild carriers), are being reduced in number by killing and the dogs are vaccinated.

This is somewhat similar to the situation which developed in the Caribbean after the mongoose was introduced there to help

eliminate the island's vast number of snakes. They became infected with rabies and explosive epizootics of the disease ravaged Grenada and Puerto Rico until the mongoose population was reduced.—*Pan American Sanitary Bureau, News release, April 3, 1959.*

Feline Practice Tips

In a practice in which there is not sufficent demand for cat blood to bank it, it is advisable to keep a strong healthy donor. The donor is anesthetized and the blood is taken directly from the heart by means of a syringe and needle rinsed in heparin or citrate solution. A ½-inch, 25-gauge needle is used to transfuse the blood into the recipient. Cats' veins are most easily entered with a needle of this size and length, and the small gauge also has the merit of preventing too rapid administration of blood.

• • •

When feline infectious anemia is suspected, it is especially important to rinse the stained blood film thoroughly. Precipitated stain is often responsible for an incorrect diagnosis, because the particles of stain can be mistaken for infectious anemia bodies.

• • •

Unless erythrocyte counts are done frequently and regularly in a laboratory, and by a skilled technician, there are tremendous chances for error. For this reason, except in occasional special cases, we have for some time relied on the hemoglobin, or better still, the hematocrit reading for information formerly supplied by counting.—*Jean Holzworth, D.V.M., Boston, Mass., at 1959 Meeting of the Alabama V.M.A. at Dothan.*

Intrauterine Infection with *Toxocara Canis*

When 7 pregnant bitches were given 20,000 infective ova of *Toxocara canis*, intrauterine infection resulted in every case. Surgical removal of fetuses at intervals during gestation demonstrated that (1) invasion of the fetus did not occur before the forty-second day of development, and (2) that the larvae required at least 14 days following infection of the bitch to invade the tissues of the uterus and fetus.

Five of the bitches, kept free of additional exposure to *T. canis*, were rebred. When they whelped, 241 to 358 days after the initial infection, patent *T. canis* infections developed in every litter within 23 to 40 days (av. 31.8) and in every bitch within 25 to 46 days (av. 32.0). These infections persisted in the bitches 9 to 108 days (av. 60.0) and were aborted spontaneously as determined by fecal examination.—*J. R. Douglas, Ph.D., and N. F. Baker, D.V.M., Ph.D., Davis, Calif., at 1959 Midwinter Conference of California V.M.A., Davis.*

Congenital Cataract in the Dog

Of 1,129 Beagles raised under identical conditions, 1 had complete bilateral developmental cataracts at 5 months of age. Of 25 pups sired by this dog, 22 had cataracts. In the eyes of pups with complete cataracts, the outstanding lesions were microphthalmia, retinal folds, and opacity of the lens. Pups with partial cataracts had only slight microphthalmia and retinal folding.

This form of cataract may best be explained as a defect in the development of the secondary lens fibers apparently resulting from incomplete continuity of the crystalline lens. Organs and skulls from all pups were examined but ocular defects were the only lesions found.—*Vet. Bull. (March, 1959): Item 896.*

Cataracts Induced in Pigs

The incidence of senile cataract is higher, and the age of onset lower, where protein malnutrition is prevalent, as in India. When young pigs were given rations containing 4.5, 6.5, and 10.0 per cent of protein, their growth rate was much lower than that in control pigs given a 20 per cent protein ration.

Cataracts appeared in pigs on the lowest protein ration after about three months. The biochemical and histological features of these lenses resembles those in rats deficient in methionine. Sunlight or ultraviolet irradiation may aggravate the condition.—*J.Am.M.A., 168, (Dec. 27, 1958): 2293.*

Toxicity of Strongylus Larvae

When larvae of *Strongylus vulgaris* died in horses, the decomposition products could be sufficiently toxic to cause signs of colic.—*Vet. Bull. (March, 1959): Item 773.*

Surgery and Obstetrics
and Problems of Breeding

Pneumoretroperitoneum, a Radiographic Technique in the Dog

WILLIAM D. CARLSON, D.V.M.

Fort Collins, Colorado

FOR RADIOGRAPHY, pneumoretroperitoneum is accomplished by injecting gas into the retroperitoneal space as a contrast medium. The retroperitoneal area is the subserous cellular tissue filling all the spaces around the peritoneal cavity and within the folds of the peritoneum.

This study was conducted concurrently with the study of pneumoperitoneum in the dog that has been reported previously.[2] Pneumoretroperitoneum in human medicine had its beginnings in its present form in 1948.[8] Since that time, it has nearly replaced pneumoperitoneum.[1,3-12] With the exception of one group of physicians who used pneumoretroperitoneum experimentally in dogs,[10] no mention has been made of its use in animals in the available veterinary literature. The objective of this study was to test its suitability for dogs.

Fig. 1—Injection site for pneumoretroperitoneum in a dog.

From the College of Veterinary Medicine, Colorado State University, Fort Collins. Dr. Carlson is the radiologist at the College of Veterinary Medicine.

The author thanks Dr. William V. Lumb, College of Veterinary Medicine, Michigan State University, for his assistance in this study.

PROCEDURE

The dogs used in this study differed in age, breed, and sex. Preparation of the dogs included withholding food and water for 12 hours prior to radiography. In addition, a suitable cathartic was given to empty the gastrointestinal tract. Only morphine sedation was used before the injection of gas.

The technique for the injection of gas was selected after many methods had been investigated. The site was 1½ inches anterior to the anus, midway between the fourth coccygeal vertebra and the ischium (fig. 1). An 18-gauge needle was inserted on a horizontal plane inward at an angle toward the midline. The needle was directed, by an index finger in the rectum, to a point ¼ inch below the ventral surface of the sacrum. The injection apparatus consisted of a large glass syringe attached to an Ayer's three-way valve. The valve was attached to a 2-ft. section of rubber infusion tubing which had an adapter for the injection needle.

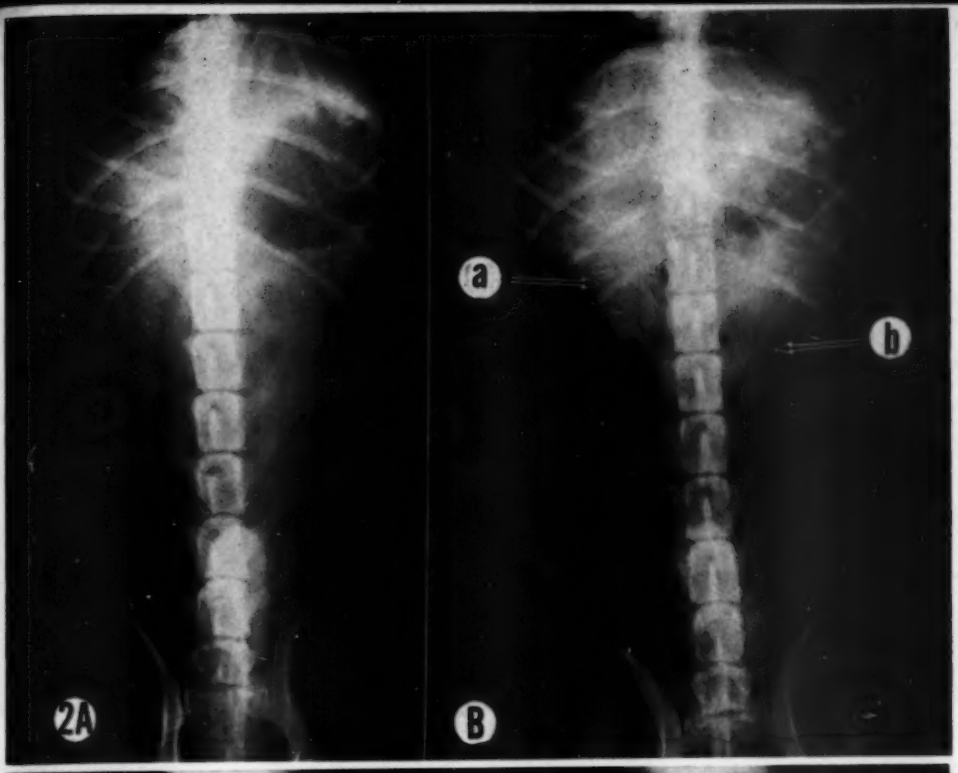
The gases used for this study were atmospheric air and oxygen. The amount of gas injected in each instance was 300 cc. The dogs varied in weight from 14 to 24 lb. The positions used were dorsoventral, ventrodorsal erect, lateral erect, and standing lateral.

Radiographs were taken at a constant machine setting of 75 kv.p. and 15 ma.; time was the only variable. Anode-film distance was constant at 30 inches.

Legends for Figures on Opposite Page

Fig. 2—Radiographs of the abdomen of a dog in the dorsoventral position—ordinary noncontrast radiograph (A); pneumoretroperitoneum (B); (a) right kidney, (b) left kidney.

Fig. 3—Radiographs of the abdomen of a dog in the ventrodorsal erect position—ordinary noncontrast radiograph (A); pneumoretroperitoneum (B); (a) right kidney, (b) left kidney.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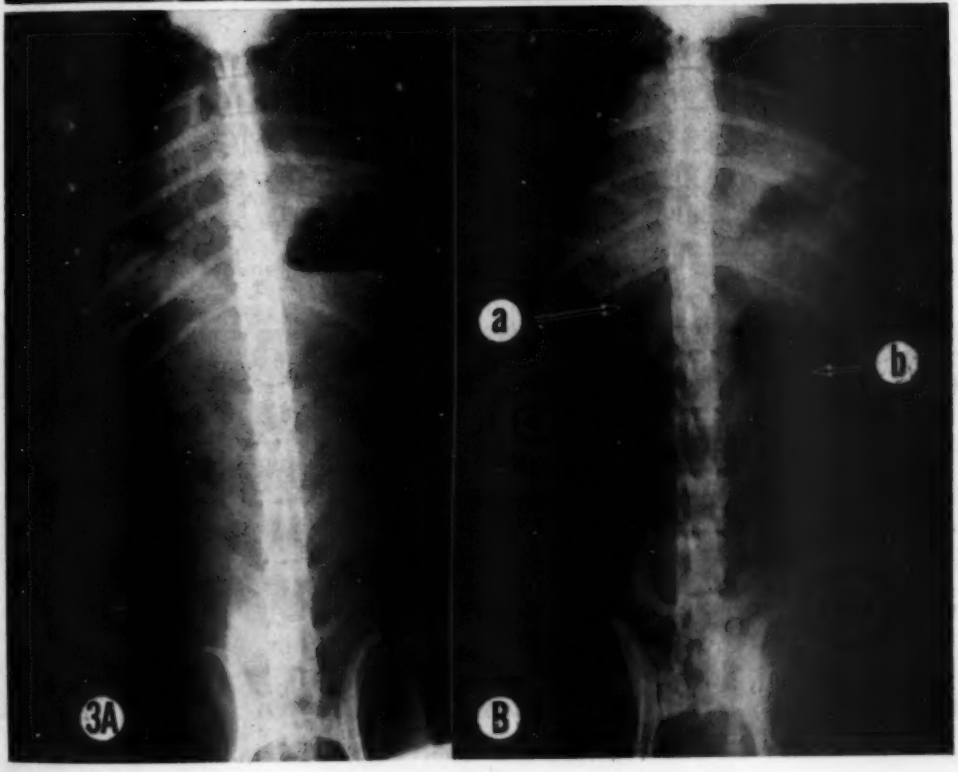
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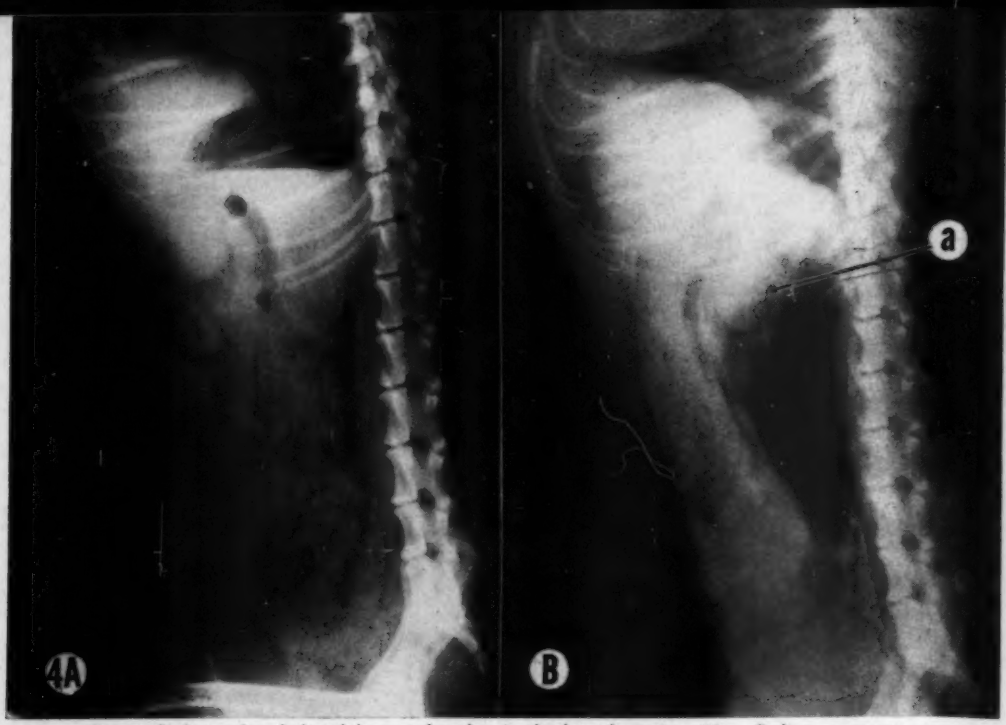

Fig. 4—Radiographs of the abdomen of a dog in the lateral erect position. Ordinary noncontrast radiograph (A); pneumoretroperitoneum (B); (a) kidneys.

RESULTS

In developing this technique, air was inadvertently injected into the middle sacral artery or vein, causing a fatal air embolism in 3 animals. Injection of the gas into the retroperitoneal space was accomplished in 8 of the 10 dogs in the study. The gas was found in the desired bilateral position in only 4 of the 8, and was on only one side in the other 4. In 2 dogs, part of the gas escaped into the peritoneal cavity.

Morphine sedation failed to alleviate all signs of pain. Air and oxygen were used with no apparent difference in visualization. In this procedure, absorption of both gases was prolonged. Oxygen was absorbed completely in 24 days; air was absorbed in 32 days. The most satisfactory positions for this technique were the dorsoventral,

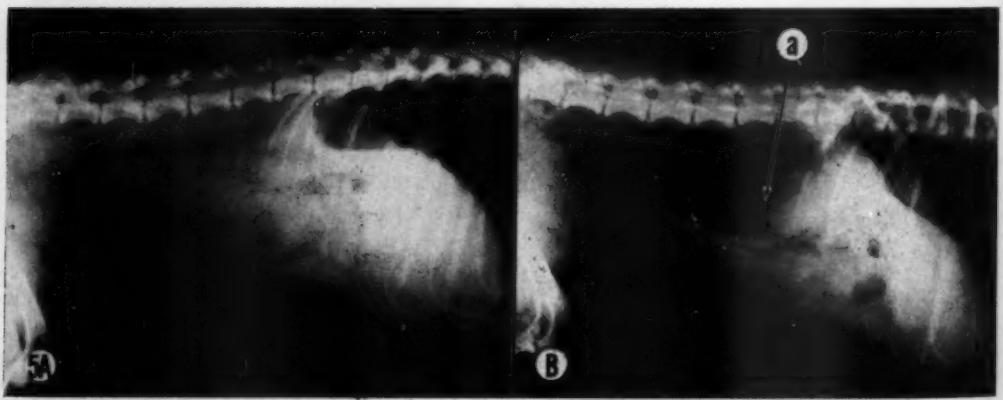

Fig. 5—Radiographs of the abdomen of a dog in the standing lateral position. Ordinary noncontrast radiograph (A); pneumoretroperitoneum (B); (a) kidneys.

using a vertical x-ray beam, and the standing lateral position, using a horizontal x-ray beam.

The criterion for evaluation of this technique was limited to the identification of the six lobes of the liver and the two poles of each kidney. The 2 dogs in which gas escaped into the peritoneal cavity were not considered in these results. Of the 8 remaining animals, 5 had both right and left kidneys clearly outlined, 2 had only the right kidney outlined, and 1 had the right kidney and the posterior pole of the left kidney outlined (fig. 2-5). Pneumoretroperitoneum failed to assist in the visualization of the liver.

Before injection of gas, radiographs of the same 10 dogs outlined the left kidney in 4 and the right kidney in only 1.

Necropsies at the termination of the study revealed no significant lesions.

DISCUSSION AND CONCLUSIONS

Development of a satisfactory technique for pneumoretroperitoneum was almost impossible as far as consistent results were concerned. In order to avoid fatal air embolism, an 18-gauge needle had to be suspended freely in the area ventral to the sacrum to avoid the vessels adherent to the ventrum of the sacrum. The rectum was occasionally punctured in positioning the needle. This constantly presented a hazard which was not solved. In light of this work, the published radiographs of pneumoretroperitoneum in experimental dogs[10] were probably pneumoperitoneum inadvertently injected.

Pneumoretroperitoneum, when successful, is an excellent method of visualizing the kidneys. As used in this study, however, it was found not to be a practical radiographic technique in veterinary radiology.

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Blood for Transfusions in Dogs

In dogs there are at least five distinct blood groups: A, B, C, D, and E.

It is only the A factor which causes concern; groups B, C, D, and E seem to produce no antibody likely to cause a great deal of trouble on transfusion.

An initial transfusion causes little trouble, as strong isoagglutinins do not occur naturally. It is only after the patient has been sensitized by an initial transfusion that reactions occur.

An anti-A producing factor is present in approximately 60 per cent of random dogs and is obviously significant where continuous transfusion is contemplated.

It is important to impress upon the owners of transfused dogs that they should not have a subsequent transfusion elsewhere without warning the veterinary surgeon in charge.—*E. M. Pittaway, M.R.C.V.S., Coventry, England, at 1958 First Annual Congress of British Small Animal Veterinary Association at London.*

Ability of a Fetus to Withstand Anoxia

When the umbilical cord was ligated in fetal lambs at 83 to 91 days of gestation, their blood pressure and heart rate were lowered, but they would survive up to 40 minutes without permanent damage. Lamb fetuses of 120 to 146 days survived only 15 to 20 minutes of this anoxia.—*Vet. Bull. (March, 1959): Item 851.*

Clinical Data

The Relationship of Hepatitis X of Dogs and Moldy Corn Poisoning of Swine

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IN 1952, AN EPIZOOTIC of canine hepatitis, later designated as "hepatitis X," was reported.[3] In 1955, this initial and two succeeding outbreaks were reviewed, and it was reported that the disease could be reproduced by feeding certain lots of a brand of commercial dog feed which was being fed to most of the dogs naturally affected during the third outbreak.[2]

In 1953, a moldy corn poisoning of hogs and cattle was described.[4] This condition occurred in swine on fields of early soft corn and was first observed during August to November, 1952. It has since been observed rather frequently in a number of states and under essentially the same circumstances as in the first report.

Several characteristics of these two entities were similar: (1) Icterus was frequently present, except in the more acute cases of the entity in swine; (2) grossly and microscopically, the liver showed a variable amount of fatty change with cirrhosis in the more chronic cases; (3) the necrosis of the liver cells was followed by marked regeneration and hypertrophy; (4) there was considerable proliferation of bile ducts; and (5) terminal hemorrhages occurred in a variety of organs, tissues, and cavities.

These similarities (previously emphasized[4]) and the inability to transmit hepatitis X of dogs[3] indicated the possibility of a common etiological factor. Therefore, moldy corn was supplied for experimental use* from a field in southern Georgia where swine had died of a condition diagnosed by the practicing veterinarian as moldy corn poisoning, on the basis of characteristic history, signs, and lesions. This corn was shelled, ground, and fed to experimental dogs. Shortly after the start of the feeding tests, the dogs began to refuse the mixture of equal parts of dehydrated dog meal and ground moldy corn. Thereafter, the corn was given in capsules daily until the death of the dogs.

It was demonstrated in these tests, the details of which will be reported later, that the ground corn was consistently toxic for dogs and produced a condition indistinguishable grossly and microscopically from hepatitis X.

This paper reports confirmed moldy corn poisoning in a herd of swine, with subsequent experimental production of a toxic hepatitis indistinguishable from hepatitis X in a dog fed ground corn from that farm.

MOLDY CORN POISONING IN SWINE

On Oct. 30, 1958, a shoat, 6 months old, was submitted for diagnosis of a condition affecting a herd of about 40 swine on a farm in southeastern Alabama. The owner reported that the condition had been present for about two months, that 6 had died, and that many others were showing poor growth. The ration consisted of ground feed made from home-grown corn mixed with a supplement. The affected swine had been fed a variety of early corn which had been exhausted by the time the shoat was presented for diagnosis. The owner stated that some of the corn was moldy. He also stated that during the time of the trouble several dogs on the farm had died after "turning yellow" and passing considerable blood in the feces.

The shoat showed dyspnea and pale mucous membranes but had a normal temperature. The significant gross lesions were: generalized icterus; abdominal cavity filled with blood; massive hemorrhage around both ovaries; and a mottled, yellow liver which was quite firm. On the basis of history and gross lesions, a diagnosis of moldy corn poisoning was made.

Microscopic examination of the liver showed that the normal architecture was absent, and the parenchymatous cells were

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*Through the courtesy of Dr. W. L. Sippel, Georgia Coastal Plain Experiment Station, Tifton, Ga.

arranged in small round groups. The cytoplasm was granular, and many cells contained vacuoles characteristic of fatty change. Special fat stain demonstrated the presence of large numbers of fat globules corresponding to these vacuoles in the sections stained with hematoxylin and eosin. There was generalized proliferation of the interlobular connective tissue with extension into the lobules. Many accessory bile ducts were present within the lobules and in the interlobular connective tissue. Some lymphocytes and polymorphonuclear leukocytes were found in the area of the central veins. Small areas of hemorrhage were scattered throughout the tissue section.

The kidney showed atrophy of some glomeruli and generalized degeneration of the tubular epithelium characterized by swelling and slight fatty change.

The history, clinical signs, and gross and microscopic lesions of this case were essentially the same as those previously described.[4]

The owner agreed to supply, for feeding tests, the available feed that had been prepared from the early corn. This consisted of only 35 lb. of ground feed prepared for chickens on the farm. No information was given regarding the other constituents in the feed, but it appeared to be mostly corn.

Two molds were isolated from this feed —one of the genus Aspergillus and the other of the genus Penicillium. No specific identification has been made.

Toxicity Test in a Dog

A 20-lb. dog, about 6 months old, that had been raised on commercial dehydrated dog meal was given this feed mixed with equal parts of the dog meal beginning on Nov. 12, 1958. From that time until he refused to eat this mixture, on December 6, the dog consumed approximately 9¼ lb. of the "toxic feed." Approximately ¾ lb. of the feed was then administered in capsules, between December 8 and 15 when the dog died, making a total of 10 lb. during 34 days. Shortly before death, the dog showed a slight icterus and began passing blood-stained feces.

The gross lesions were generalized icterus, marked hemorrhage into the lumen of the small intestine, and severe fatty change of the liver.

On microscopic examination, the parenchymatous hepatic cells contained small vacuoles giving the foamy appearance characteristic of fatty change. The cytoplasm stained lightly; nuclei were small and dark-staining. Bile duct proliferation was prominent in the interlobular connective tissue, and in many areas the hepatic cells appeared to be forming clusters of bile ducts. Small areas of regenerating hepatic cells with dark-staining cytoplasm were found near the periphery of the lobules. There was slight infiltration with lymphocytes and polymorphonuclear leukocytes.

The gross and microscopic lesions were indistinguishable from those of the cases of hepatitis X observed previously and produced experimentally by feeding toxic dog feed.

Discussion

On the basis of these observations, and those to be reported in more detail elsewhere, it is concluded that hepatitis X of dogs and moldy corn poisoning of swine are caused by the same etiological factor. In addition to the similarities of these conditions, it is of interest that the seasonal occurrence of the natural disease is consistent with the above conclusion. Moldy corn poisoning in this area has been most frequently observed during the late summer and early fall, while hepatitis X has occurred during the months of November to April (1951-1952), September to January (1952-1953), and October to February (1954-1955). Certain lots of commercial dog meal manufactured during these periods could have been made of ingredients containing some moldy corn of these early maturing corn varieties.

It is of interest also that molds of the genus Aspergillus and Penicillium were isolated from the feed used on affected farms (13 cultures of mold were recovered from corn taken from fields in Georgia where hogs were dying).[1] Two of these, *Penicillium rubrum* and a strain of *Aspergillus flavus* (Link), proved toxic when grown in pure culture on autoclaved corn, dried, ground, and fed to swine.

It is possible, of course, that other molds, and even other toxic agents, might produce conditions similar to these two entities or that the definition of these entities has been too restricted. During the past six years, we have observed a small number of cases in the routine postmortem examinations of dogs, in the Department of Pa-

thology and Parasitology, in which the gross lesions were essentially identical to those of hepatitis X, but which did not show the characteristic microscopic changes.

SUMMARY AND CONCLUSIONS

A case report of moldy corn poisoning in swine is presented. Feed produced from the same corn was toxic when fed to a dog for 34 days. The lesions produced in the dog were indistinguishable from those of natural and experimental cases of hepatitis X.

It is concluded that hepatitis X of dogs and moldy corn poisoning of swine are produced by the same etiological factor, which appears to be a toxin or toxins produced by certain molds.

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The Treatment of Hog Cholera with Specific *Gamma* Globulin

Ten susceptible pigs weighing 15 to 16 kg. were inoculated intramuscularly with 0.25 ml. of virus. Fort-eight hours later, 5 of these pigs were given 4 to 6 Gm. of specific *gamma* globulin. This dose was the equivalent of 100 to 150 ml. of antiserum. It was dissolved in physiological saline solution 1:10 just before use. The pigs had fever and anorexia when treated, but recovered after a mild course of hog cholera. When challenged 43 days after infection, they were immune to 0.5 ml. of virus given subcutaneously.

Three pigs treated four days after infection showed the typical hog cholera syndrome and 1 died after 11 days; 2 recovered. The 2 untreated controls died in six days with signs and lesions of cholera.

In another experiment, 12 pigs were inoculated subcutaneously with 0.5 ml. of virus. Two untreated controls died of cholera. The remaining 10 pigs were given two doses of 20 to 22 Gm. of *gamma* globulin at 24-hour intervals. The 2 pigs treated 24 hours after infection had a maximum temperature rise of 0.6 C. and no loss of appetite. The 4 pigs treated during the first marked rise of temperature recovered after a light illness, with 1 exception; it required three doses of globulin. The 4 pigs treated 24 hours after the rise in temperature recovered—1 after a severe illness. Three susceptible pigs kept with the others contracted typical cholera. Two were treated when they became ill and they recovered. One left untreated died in six days.

The authors conclude that *gamma* globulin is an effective treatment if given within 24 hours after the first rise in temperature.—[*P. Ya. Shcherbatykh et al.: The Treatment of Swine Sick with Hog Cholera by Means of Specific Gamma Globulin. Veterinariya, 36, (Jan., 1959): 36-40.*]—ROBERT E. HABEL.

Cultivation of Immunological Variant of Hog Cholera Virus

Whether procedures could be developed whereby the immunological variant of hog cholera virus responsible for the losses of swine in the Midwest in 1949 could be isolated, was investigated. The methods used proceeded from the assumption that the simultaneous virus used in vaccination was a mixture of regular and variant viruses.

Since current studies had shown that regular virus could be propagated in tissue culture medium, it seemed possible that this method, with modifications, might also be used to separate the variant virus in a stable form, with retention of its identifying characteristics.

Previous investigations had shown that it was necessary to make serial passages of the variant virus in swine simultaneously with a subprotective dose of serum in order to maintain a virus capable of showing immunological variation.

Earlier work had shown that the mixture of regular virus and variant virus retained characteristics after 16 passages in swine —the first nine passages simultaneously with subprotective doses of serum and the last seven without serum. However, the variant characteristics were lost after

seven more serial passages without serum. Consequently, starting with the sixteenth passage, 12 additional passages were made. The first seven passages were made with serum and the last five without serum, making a total of 28 passages. When tests in swine showed that the twenty-sixth passage had characteristics of variant virus, the twenty-eighth passage was used as inoculum for the first passage in tissue culture. The tenth serial passage in tissue culture, when tested in swine, still retained its variant characteristics.

If the virus continues to maintain its stability under these procedures, it will provide new approaches to viral studies of hog cholera.—[*C. N. Dale and J. R. Songer: In Vitro Propagation of Hog Cholera Virus. III. Cultivation of An Immunological Variant, with Retention of Its Identifying Characteristics. Am. J. Vet. Res., 20, (March, 1959): 311-318.*]

African Swine Fever in Portugal

In 1957, a highly contagious viral disease caused mortalities up to 100 per cent of swine herds in Portugal. The only means of control was eradication by the "stamping out" (herd slaughter) method. The virus was serially transferred on swine testicular tissue but no attenuation of its original virulence was obtained.—*J. M. Ribeiro et al. in Off. internat. des Epizoot., 50, (May, 1958): 516.*

Subclinical Teschen Disease

Blood samples from 148 swine in 92 herds, of two communities in Germany, were serologically tested for Teschen disease. Although the disease had been observed in only six of the 92 herds, the serums of 13 animals from nine herds were clearly positive and another 33 were suspicious. Only 1 of these 46 animals later developed the clinical disease, and in only one of the infected herds did the disease develop in other swine.—*F. Hecke in Monatsh. f. Tierheilk., 2, (1959): 33.*

Torsion of Liver Lobes in Sows

Over a period of three years in a herd of 2,000 sows, 4 aged sows, each with a large litter, developed liver torsions during their third week of lactation. Either the right or left outer lobe was rotated up to 360 degrees. In 1 sow, the liver capsule was ruptured and there was severe hemorrhage. All showed extensive peritonitis with blood-tinged fluid.

The sows first showed inappetence and restlessness, followed by severe, prolonged vomiting. All died in 12 to 18 hours.—*Vet. Bull. (March, 1959): Item 825.*

Pasteurellosis in the Horse

The horse, as well as other animals, can be a carrier of pathogenic pasteurellas and remain healthy until its resistance is disturbed. Horses used for the production of serums, such as tetanus antitoxin, may develop clinical pasteurellosis following injections of antigens. Treatment with anti-Pasteurella serum, sulfanomides, and antibiotics have been ineffective.—*Vl. Carabulea in Scientific Papers from Pasteur Serum and Vaccine Institute, Bucharest, 3, (1958): 529.*

Experimental Ornithosis in Turkeys

The path of toxigenic ornithosis virus through turkeys infected by air-borne and oral exposure was traced by examination of their tissues. Those exposed to the aerosols became acutely affected and 15 per cent died. High concentrations of the virus were found in their lungs and air sacs within 24 hours and in the blood stream within 48 hours. Concentration of virus in the pericardium increased gradually. Tissues of those which survived apparently became free of the virus in about two months.

Birds exposed orally showed no clinical effects or serological response, but they excreted the agent and infected other birds. Thus, ornithosis may be initiated in a few birds by ingestion and may spread rapidly when aerosols containing their desiccated infected excreta are created.—*L. A. Page in Avian Dis. (Feb., 1959): 51.*

Inactivating Ornithosis Virus

Turkeys with ornithosis, which had been processed and frozen, contained viable virus after 372 days of storage at -20 C. (-4 F.). In a 20 per cent mammalian tissue suspension, the virus was destroyed in five minutes at 56 C. (132.8 F.), and in 48 hours at 37 C. (98.6 F.). It is killed quickly at cooking temperatures.—*L. A. Page in Avian Dis. (Feb., 1959): 67.*

Promazine in Canine Medicine

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OF THE MANY problems facing the veterinarian in small animal practice, two are most frequent: (1) control of the animal for physical examination and minor therapeutic procedures, and (2) the pre- and postoperative care of the animal undergoing major surgery.

Physical restraint for general examination or minor procedures usually includes muzzling or, if the animal is readily controllable, the owner may restrain the subject. When examination of the mouth and throat is necessary, leather straps, looped strings or tapes, or a speculum may be used. There are animals, however, whose hyperexcitability precludes adequate examination by use of these simple methods of restraint.

During preparation of the animal for surgical procedures, physical restraint may be necessary. Postoperatively, it is even more important because of the possibility of self-inflicted damage during recovery from anesthesia. Also, sutures and bandages are subject to multilation by the animal.

Until recently, the use of hypnotics and sedatives provided the only recourse for the control of hyperexcitable animals. Unfortunately, these agents sometimes produced undesirable side effects by their depression of the higher cortical centers. The development of a series of ataraxics has provided several useful active compounds without these disadvantages.

Rauwolfia, obtained from the root of the shrub *Rauwolfia serpentina*, was used in India and neighboring tropical countries for centuries in the treatment of a variety of human disorders, especially certain types of insanity. Recently, the drug has attracted more attention since the isolation of a number of active alkaloids and the demonstration of their effects on the cardiovascular system. Currently, Rauwolfia derivatives are receiving attention as antihypertensive agents and as mood-modifying agents in psychiatric patients. One of the Rauwolfia derivatives, reserpine, is an effective ataractic agent for restricted veterinary use.[2,3,8,9]

Meprobamate (Equanil*) is a longer-acting propanediol derivative. The drug has both muscular relaxant and anticonvulsant properties, and is virtually free from side reactions. Although meprobamate is effective in the control of hyperexcitable, nervous, or unruly animals, its poor solubility in water requires that it be given orally.[4,5]

Several phenothiazine derivatives, by their alteration of metabolic functions of the cells of the autonomic nervous system, reduce the aggressive response to environmental circumstances. These compounds have been used to facilitate handling and to encourage cooperation of the subject during clipping and bathing, roentgen therapy, etc.[1,2,3,9,11] These drugs, used before or during the induction of anesthesia, make possible a considerable reduction in the amount of anesthetic.[11]

Chlorpromazine, one of the phenothiazine derivatives, is well tolerated by animals and provides effective chemical restraint, but is reported to be contraindicated in animals with severe depression of the central nervous system and in those with extensive liver damage.[10] In addition, other side effects may occur when it is used in conjunction with other agents.[1]

Promazine (Sparine**), another member of the phenothiazine series, reduces response to disturbing stimuli and thus produces tranquillity without depressing the cortical centers. Less depression and lethargy result from the action of promazine than from chlorpromazine, and recovery is more rapid.[7]

The use of promazine for the control of central nervous system excitation, anxiety, and agitation has brought considerable change in the management and treatment of animals.[2,5-8] It may be administered orally or parenterally. It exerts a quieting influence without marked hypnosis; it obviates the pronounced depressive activity and

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*Equanil is produced by Wyeth Labs., Philadelphia, Pa.
**Sparine is produced by Wyeth Labs., Philadelphia, Pa.

side effects of earlier related compounds. In surgical procedures, in conjunction with a general anesthetic, it eliminates the excitement stage; the postoperative period is less "stormy," recovery time is considerably shortened, and the amount of general anesthetic required is markedly reduced.

CASE REPORTS

In the treatment of 249 dogs, we have found promazine to be highly effective. The following clinical reports are examples of its practical application.

1) When dogs not used to confinement are hospitalized, they become nervous, noisy, or even vicious. Promazine, administered intramuscularly to 125 dogs at the rate of 2 to 3 mg. per pound of body weight, once or twice daily, resulted in easier handling, improved appetite, and a quieter hospital.

2) For a nervous dog or one difficult to handle during physical examination, promazine is given intramuscularly. After ten to 15 minutes it is usually tractable. Thirty-five dogs were successfully handled in this manner.

3) In the treatment of conditions such as dermatitis, otitis, and arthritis, promazine tablets are dispensed to be given at the rate of 1 to 2 mg. per pound, three times daily, in conjunction with other specific medication. Fifteen dogs treated in this manner were more at ease, had improved appetite, and responded more satisfactorily to basic medication.

4) In 30 dogs, the injection of promazine before the administration of vermifuges prevented nausea and abdominal cramps.

5) Many long-haired dogs presented for clipping and grooming become upset at the sound of electric clippers. Promazine, given intramuscularly to 10 dogs at the rate of 2 mg. per pound 15 minutes before handling, calmed them sufficiently to permit grooming. Occasionally, a highly excitable animal must be given thiopental sodium, intravenously, to obtain the desired control.

6) Promazine used in conjunction with local anesthetics for minor surgical procedures (removal of small growths or cysts, suturing traumatic wounds, dental work) eliminates the need for a general anesthetic. Surgery was readily performed on 18 dogs in this category; they remained quiet and their immediate release was possible.

7) After recovery from a general anesthetic, promazine injections are given (2 mg. per lb. once or twice daily) while the animal is hospitalized. Promazine tablets are dispensed for home use at the rate of 1 mg. per pound two or three times daily until the sutures are removed. Sixteen dogs for which this regimen was prescribed had more rapid healing.

A dose of 2 mg. per pound, given intramuscularly, brings optimal results within 20 minutes. In some highly agitated animals, the dose has been increased to 3 mg. per pound, given intramuscularly, or 4 mg. per pound, given orally, to obtain the same results.

Promazine also was used in the treatment of domestic cats and 1 ocelot at the same dosage levels with varying results.

CONCLUSIONS

Promazine therapy, given to 249 dogs to facilitate examination, to support local anesthetics for minor surgery, or to precede general anesthetics, proved to be valuable in veterinary practice. The animal benefits by its use, the client is pleased, and the veterinarian may work effectively.

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A Calcium Lactate-Aluminum Hydroxide Preparation as a Preventive for Parturient Paresis

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UNTIL RECENTLY, no relationship had been established between the ration fed to the dairy cow and the development of parturient paresis. In 1951, it was demonstrated that feeding high levels of vitamin D for three to eight days prepartum was effective in preventing this disease.[6] In 1954, the disease was reported to have been prevented by feeding a low-calcium, high-phosphorous diet for about one month prepartum.[1]

In 1952, cows which ultimately were affected with parturient paresis were found to be in a severe negative calcium balance for at least two weeks prior to calving.[9] This finding would seem to indicate the desirability of a feeding regimen which would increase calcium absorption prior to parturition.

The present experiment was designed to study the efficacy of a product, Paracalcin,* containing a combination of calcium lactate and aluminum hydroxide. Such a product has been shown to improve calcium retention and reduce neuromuscular irritability in pregnant women.[7] This treatment produced an increase in calcium and a decrease of phosphorus in the blood serum.

Calcium lactate as a complex should be more readily absorbed than most calcium salts. Aluminum hydroxide has a strongly adsorptive surface for anions, such as phosphorus, at the pH normally occurring in the digestive tract.[2] It has been shown that when chickens are fed large amounts of reactive aluminum hydroxide gel, a severe rickets is produced which can be largely reversed by the injection of phosphate salts.[8]

EXPERIMENTAL PROCEDURE

This study involved 19 cows in the Colorado State University dairy herd. The age, breed, treatment, and past history of parturient paresis for these cows are presented (table 1). All but 2 of these cows had been affected at least once. These cows (36 and 72 [1957]) were included because they seemed to be likely candidates for the disease because of age and high production.

The drug administration was started an estimated eight days prior to parturition, but the usual difficulty was encountered in predicting the day of calving, and the length of treatment ranged from two to 26 days. In two cases, treatment was stopped when it seemed apparent that the cow would not calve on the estimated date. The product was given either in a gelatin capsule or by drench, once daily, in order to assure consumption of an exact amount by each cow.

Every cow in the University herd which had previously been affected was treated. Controls of untreated cows were not used because this would have unduly restricted the number of treated animals. It seemed that fully as good information could be obtained by treating all animals which developed parturient paresis, and comparing the incidence of the disease with its repeatability. A repeatability of 50 per cent had been observed in the Ohio Experiment Station herd.[5] The repeatability in the Colorado State University herd appears to be higher than this, although no exact figures are available.

Blood samples were taken from the jugular vein daily prior to treatment until calving. Whenever possible, a blood sample was collected immediately after calving. Also, if the cow showed parturient paresis, a sample was collected prior to treatment with intravenous calcium gluconate. Blood serum analyses from these samples were made for calcium and phosphorus. Calcium was determined by titration with ethylenediaminetetraacetate (Versene**)[10]

From the Department of Dairy Industry, Colorado State University, Fort Collins.

The authors acknowledge the assistance of Dr. Robert Pierson and Dr. B. G. Erwin, Ambulatory Clinic, Colorado State University, who were in charge of herd health for the University dairy herd and helped conduct this experiment. They also thank Allen Lamb, Windsor, Colo., and Joe Willett, manager, Sinton Dairy Farms, Colorado Springs, for conducting the field trials for this experiment.

*Paracalcin was furnished for this study together with grant-in-aid funds by Warner-Chilcott Laboratories, Morris Plains, N.J.

**Versene is produced by Fisher Scientific Co., Fairlawn, N.J.

after checking this method against the method of Clark and Collip.[3] Phosphorus was determined by the method of Fiske and Subbarow.[4]

The calcium-aluminum compound was given at the rate of 30 Gm. per day. This product was supplied in two forms; one contained about 500 I.U. of vitamin D_3 per daily dose; the other was the same except it contained no vitamin D.

In addition to the cows in the University herd, these two types of the compound were fed to mature cows in two other herds in Colorado. One herd consisted of about 80 purebred Jerseys; the other of about 300 grade Holstein-Friesians. In these herds, the product was fed in the grain ration for an estimated eight days prior to parturition. No blood samples were analyzed from these herds.

RESULTS AND DISCUSSION

Of the 19 treated cows in the University herd, 13 did not develop parturient paresis but 6 did. However, cows 72 and 55 had been treated for only two and four days, respectively. The 2 cows treated for the longest periods (15 and 26 days) both developed parturient paresis. Treatment ranged from six to 11 days for the cows which did not develop the disease.

TABLE 1—Treated Cows Which Did Not Develop Parturient Paresis

Cow No.	Breed*	Age (yr.)	Parturient paresis history (No. of attacks)	Days treated
3	H	10	2	10 (D)
99	H	8	2	6 (D)
108	J	6	2	11 (D)
70	G	9	3	7 (D)
43	H	6	2	6 (O)
89	J	5	2	11 (O)
15	H	9	2	8 (O)
79	J	9	4	7 (O)
50	G	12	6	8 (D)
101	H	8	1	7 (O)
107	H	7	2	10 (O)
18	H	7	1	6 (O)
72 (1957)	H	11	1	6 (D)
Treated cows which ultimately developed parturient paresis				
72 (1958)	H	10	none	2 (O)
36	H	6	none	15 (D)
55	H	11	2	4 (O) (R)
109	H	5	1	8 (D) (R)
103	G	7	2	26 (D) (R)
19	H	11	3	12 (D)

*H = Holstein-Friesian; J = Jersey; G = Guernsey.
D = Paracalcin containing vitamin D; O = Paracalcin without vitamin D; R = relapse of parturient paresis and treated a second time.

If the 2 cows without a previous history of parturient paresis are eliminated, 4 of 17 treated cows (25%) were repeaters. The additional vitamin D did not improve the effectiveness of the product (table 1).

The data on blood serum calcium and phosphorus are presented for 4 representa-

TABLE 2—Blood Serum Calcium and Phosphorus Levels of Treated Cows Which Did Not Develop Parturient Paresis

Date	Ca(mg./100 cc.)	P(mg./100 cc.)	Date	Ca(mg./100 cc.)	P(mg./100 cc.)
Cow No. 79			Cow No. 70		
6-15 (1957)	8.8	—	1-4 (1958)	11.4	3.1
6-17	10.8	—	1-5	10.8	3.5
6-18	11.0	6.2	1-6	10.2	3.4
6-19	9.6	4.6	1-7	11.4	3.5
6-20	11.0	6.8	1-8	11.4	4.5
6-21	11.2	6.2	1-9	11.4	5.0
6-23*	10.0	2.5	1-10	10.6	3.4
6-24	10.0	4.5	1-11	10.2	3.9
			1-12*	10.2	3.8
			1-13	10.6	6.1
			1-14	10.8	5.4
Cow No. 108			Cow No. 18		
12-29	12.4	5.8	11-20	10.5	—
12-31	9.6	5.6	11-21	11.0	5.5
1-1 (1958)	10.8	4.0	11-22	10.8	—
1-2	10.8	3.5	11-23	10.4	5.0
1-3	10.2	4.4	11-24	10.4	3.7
1-4	10.2	5.4	11-25	9.6	—
1-5	11.4	5.4	11-26	9.0	—
1-6	9.6	4.4	11-27 1 a.m.*	8.8	3.4
1-7	11.0	6.2	11-27 4 a.m.	9.2	—
1-14	10.8	5.4	11-27 1 p.m.	8.8	6.6
1-15	10.8	5.0	11-28	8.6	7.4
1-16*	8.0	3.9			
1-16	9.6	4.8			
1-17	9.6	4.6			
1-18	9.9	3.9			

*Blood sample taken at time of calving.

tive cows which did not develop parturient paresis (table 2), and for 4 cows which did develop the disease (table 3).

In most cases, the blood serum calcium levels were maintained at higher levels near parturition than is commonly the case. Likewise, cows 72 and 18 developed parturient paresis while they had blood calcium levels higher than in most instances. Cows 19 and 103 had blood serum calcium levels at the time of therapy which were more typical of this disease. Blood serum phosphorus levels for both normally calving cows and for those developing parturient paresis were typical of those found in the literature for cows under similar conditions.

FIELD TRIALS

The results in two private herds indicated a marked ability of the drug to prevent the development of parturient paresis. In the purebred Jersey herd, 43 cows were treated over a two-year period; 26 had a previous history of parturient paresis, and the remainder were in an age group which is considered most susceptible. These cows were fed the product in the grain ration for three to 12 days prepartum. In this group, 5 cows developed parturient paresis; however, 2 of the 5 were fed the product for only two days prior to parturition.

In the commercial Holstein-Friesian herd, 87 cows with a previous history of parturient paresis were fed the product for a maximum of eight days. If the cow had not calved at this time, treatment was stopped. Only 1 of the 87 developed parturient paresis and she had been treated for only two days. One other cow showed signs of the disease but recovered without treatment.

In neither of these herds was there any evidence that additional vitamin D was of value as a preventive.

There is no ready explanation for the considerable difference in response to treatment between the University herd and the two private herds; however, the number of cases in the University herd was fewer and, as pointed out, 4 of the 6 cows had been treated for longer or shorter periods than any of the cows which did not develop parturient paresis.

CONCLUSIONS

The obvious conclusions from these observations are that the calcium lactate-

TABLE 3—Blood Serum Calcium and Phosphorus Levels of Treated Cows Which Ultimately Developed Parturient Paresis

Date	Ca(mg./100 cc.)	P(mg./100 cc.)	Date	Ca(mg./100 cc.)	P(mg./100 cc.)
Cow No. 72			Cow No. 18		
12-14 (1957)	10.0	.....	11-20 (1957)	10.5	....
12-15	9.8	.....	11-21	11.0	5.5
12-16	10.0	.....	11-22	10.8	....
12-17	10.2	.....	11-23	10.4	5.0
12-18	9.2	.....	11-24	10.4	5.7
12-19 a.m.*	7.8	.....	11-25	9.6	....
12-19 p.m.	8.2	.....	11-26	9.0	....
12-20	7.6	.....	11-27 a.m.	9.2	....
			11-27 a.m.*	8.6	....
			11-27 p.m.	8.8	6.6
			11-28	8.6	....
			12-6	9.2	7.4
Cow No. 19			Cow No. 103		
1-8 (1958)	11.6	6.0	11-26	10.6	4.5
1-9	11.4	5.5	11-28	9.6	3.5
1-10	10.6	5.0	11-30	12.4	3.5
1-11	10.8	5.2	12-3	10.1	4.5
1-12	11.2	4.2	12-7	10.7	5.0
1-13	11.2	4.9	12-11	10.6	5.0
1-14	11.6	5.0	12-13	10.7	4.8
1-15	11.2	6.0	12-17	11.3	....
1-16	10.8	6.8	12-19	11.3	....
1-17	10.4	6.4	1-30 (1958)	10.8	5.8
1-18	10.0	6.0	1-31	10.2	5.7
1-19 a.m.*	5.6	3.2	2-1	10.1	6.8
1-19 p.m.	4.6	2.8	2-2* 6 p.m.	6.0	2.6
1-20	6.8	6.1	2-2 10 p.m.	6.0	2.0
1-21	8.4	6.9	2-3	7.2	5.0
			2-4	8.5	5.4
			2-5	10.2	5.0

*Blood sample taken at time of calving.

aluminum hydroxide compound, when fed at the rate of 1 oz. per day, will protect a majority of mature dairy cows against parturient paresis. It may not be advisable to feed the product for more than eight to ten days prepartum, but the evidence for this is based on only 2 cows in the University herd and upon the negative evidence that in the two cooperating herds the product was never fed for more than 12 days. Likewise, these results would indicate that the product is not effective when fed for less than four days prepartum.

It was impossible to draw significant conclusions from the data on blood serum calcium and phosphorus, although there was a suggestion that the blood serum calcium levels were generally higher than normal at parturition time.

The addition of vitamin D to the product did not improve its effectiveness.

SUMMARY

Nineteen mature cows in the Colorado State University herd, 17 of which had been previously affected with parturient paresis, were treated with a calcium lactate-aluminum hydroxide product (Paracalcin) prior to parturition. Six of the 19 developed parturient paresis. Their blood serum calcium and phosphorus levels were not greatly different from those previously reported for cows suffering attacks of parturient paresis.

In two private herds, 130 mature cows with histories of parturient paresis were treated with the same product and 6 cows developed parturient paresis. The addition of 500 I.U. of vitamin D per day did not improve the effectiveness of the product.

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Crop Impaction in Pullets

J. W. FAGUE, V.M.D.

Shippensburg, Pennsylvania

A flock of 300 White Leghorn pullets, 8 months of age, had dropped in production from 250 to 150 eggs per day. A number of birds had been lost due to injuries from "cannibalism."

When the flock owner noticed some of the birds twisting and stretching their necks in an abnormal manner, he asked for veterinary assistance.

A physical examination of the birds exhibiting strange neck movements disclosed impacted crops several times their normal size. Necropsy revealed a mass of excelsior the size of a man's fist impacted in the crops.

A commercial type of excelsior nest pads, which are used in many flocks without trouble, was the source of the excelsior found in the crops. Cannibalism in the flock may have increased the birds' desire to pick up this foreign material.

The fact that these birds exhibited signs of a central nervous system disturbance made the diagnosis puzzling until a physical examination and necropsy were performed.

Dr. Fague is a general practitioner in Shippensburg, Pa.

Bovine Listeriosis in Italy

Listeria monocytogenes was isolated from a bovine fetus aborted at the seventh month of gestation. This is the first case of listeriosis in animals reported in Italy. —*Vet. Ital. (June, 1958)*: 445.

Editorial

Can Statistics Be Misleading?

The article, "Veterinary Medicine in a Changing World" (*J.A.V.M.A.*, March 1, 1959: 205), surprised old-timers who are familiar with the swine industry by stating that "comparing . . . the farm population of 1900 with that of today, we find . . . no change in the number of swine."

The source of that information was the *Statistical Bulletin* (No. 230, July, 1958, U.S.D.A.). It states that the reported number of swine on U.S. farms was 51.7 million in January, 1900, vs. 51.0 million in January, 1958.

These statistics seem convincing, but they do not indicate the total swine population. How wrong they could be is obvious when we consider that in 1900 few pigs were farrowed before April and few hogs were marketed when less than 7 or 8 months old. Thus, most of the previous year's hog crop was still on the farm and was counted in January. Today, spring pigs are farrowed earlier and most are marketed when 6 or 7 months old. Thus, in spite of the increase in fall pigs (perhaps 25% of the annual crop in 1900 vs. 40% now), a much higher percentage of the annual crop is marketed during the year of birth and is not included in the January count.

Another ready source of statistics on swine production is the amount of pork processed annually by packers. That source also is misleading because at the turn of the century few hogs were sold at less than 250 lb. Today, chiefly because of the increasing demand for "lean meat" and for smaller cuts, most hogs are marketed at considerably less than 250 lb. Thus it requires many more hogs to produce the same amount of pork.

Even so, it is estimated that pork production was 6.0 billion lb. in 1904 and over 10.5 billion lb. in 1957—an increase requiring the processing of twice as many hogs at the lesser weights.

The packer's data (acquired from Armour and Co.) indicate an increase in hogs slaughtered under federal inspection from 21.2 million head in 1895 and 29.3 million in 1900 to 60.7 million in 1957. The estimated totals for both federally inspected and farm slaughtered hogs show the annual average number as 57.7 million for the ten years prior to World War I, and 78.8 million for the ten years prior to 1958. The peak was 98.9 million in 1943.

Statistics for Veterinarians

Veterinarians usually deal with animals individually and, since their services are more often required for younger rather than older animals, the statistics which interest them most pertain to the young. This is particularly true with pigs which usually require considerable vaccinating.

Annual estimates for both spring and fall pigs which survived the hazards of the first few days of life are reported in the "Agricultural Outlook Charts" (U.S.D.A.), 1956. They show a relatively steady increase from 70.3 million pigs "saved" in 1925, the first year reported, to 100.9 million in 1955. Again, the peak (121.8 million) came in the World War II year, 1943.

Moral: Statistics are reliable only when complete or when properly selected.

Factors Affecting Pork Exports

Pork exports from the United States were 30 per cent less in 1958 than in 1957. This apparently is due to (1) fear of introducing hog cholera (swine fever) virus in refrigerated pork, (2) increased swine production in Europe (64% above 1946), and (3) inferior quality of the product (too lardy).

The danger of transmitting hog cholera virus was further reduced when five states (*see* p. 496) were added to the 18 (*J.A.V.M.A.*, Feb. 1, 1959: 145) which have restricted the use of virulent virus in vaccinating.

Recently, according to the *National Hog Farmer* (April, 1959), further remedial actions have been initiated. A congressional committee was studying the problem of producing better quality pork products, and many new swine carcass contests are being held.

The carcasses are rated chiefly on the percentage of "lean" cuts and the measurement of the "loin eye" and "back fat." In eight recent contests, the winning carcasses were from hogs weighing from 185 to 222 lb., yielding from 54.6 to 60.7 per cent of lean meat, with loin eyes 4.4 to 5.7 square inches on cross section, and back fat 1.00 to 1.37 inches deep. The great majority of winners were of Hampshire breeding.

Abstracts

Reflex Control of the Ruminant Stomach

An attempt was made to characterize certain of the nervous reflexes which originate in the ruminoreticulum—those associated with ruminoreticular motility and eructation. Eructation, deglutition, and ruminoreticular motility were recorded in adult unanesthetized cows using strain gauges activated through Brody-Quigley catheters.

Procedures used to try to locate and describe the nerve receptors associated with those reflexes included: (*a*) distention or tactile stimulation of given areas of ruminoreticular mucosa; (*b*) application of topical anesthetics to given areas of the ruminoreticular mucosa; (*c*) stimulation or anesthetization of nerves supplying the reticulum and rumen; (*d*) variation in the characteristics or quantity of ruminoreticular content; and (*e*) atropine inhibition of ruminoreticular motility.

Evidence is given which indicated that although distention of the rumen and reticulum provides a strong stimulus to their rate of contraction, eructation is associated with active contraction of the rumen. It is suggested that eructation may be initiated by volume receptors in the walls or tension receptors in the suspensory apparatus of the rumen. Evidence is also presented that the cardiac sphincter is normally in a state of tonic contraction.—[*C. E. Stevens and A. F. Sellers: Studies of the Reflex Control of the Ruminant Stomach with Special Reference to the Eructation Reflex. Am. J. Vet. Res., 20, (May, 1959): 461-482.*]

Penile Development of the Angora Goat

In the course of 400 routine necropsies, it was observed that there was nonseparation of the epithelial surfaces of the penis and prepuce in most castrated goats, while the anterior 4 cm. of the penis was free in normal males.

Microscopic examination revealed that the two layers of stratified squamous epithelium of the penis and prepuce were not separated but formed a solid band of epithelium with two opposing basal layers.

Detailed measurements on 24 animals showed that, in general, the penis of castrated males was also smaller in diameter than in normal adults.—[*Ward R. Richter: Observations on the Penile Development of the Angora Goat. Am. J. Vet. Res., 20, (May, 1959): 603-606.*]

Uterine Morphological Changes in Growing Pigs

A study of the morphological changes which occurred in the genital tracts of growing pigs between the ages of 1 day and 6 months revealed the following.

The total thickness of the endometrium increased from 126 μ in the newborn to over 5 mm. (5,520 μ) in the 6-month-old animal. It was also observed that the endometrium changed from 47 per cent of the thickness of the uterine wall in the 2-week-old pig to 69 per cent in the 4-month-old gilt.

The uterine glands, which were few in the newborn pig, developed gradually, penetrating the total depth of the endometrium to the myometrial border when pigs were 3 months old.

The mucosal folds first appeared in the 5-week-old pig and gradually increased in complexity.—[*Robert Hadek and Robert Getty: The Changing Morphology in the Uterus of the Growing Pig. Am. J. Vet. Res., 20, (May, 1959): 573-577.*]

Plaque Technique in Porcine Enteroviruses

The Dulbecco plaque technique, utilizing porcine kidney cells, was used successfully for the study of the porcine enteroviruses in: (*a*) primary isolation from feces, (*b*) obtaining pure lines from single plaques, (*c*) differentiating strains on the basis of plaque characteristics, (*d*) enumerating, and (*e*) conducting either virus- or serum-neutralizing tests.

The viruses of pseudorabies and canine infectious hepatitis, but not that of transmissible gastroenteritis of swine, produced plaques on porcine kidney cells.—[*K. V. Singh, E. H. Bohl, and J. M. Birkeland: The Use of the Plaque Technique for the Study of Porcine Enteroviruses. Am. J. Vet. Res., 20, (May, 1959): 568-572.*]

Books and Reports

Diseases of Laboratory Primates

This is the first volume of a proposed four-volume "Handbook of the Primates."

The topics are discussed on a regional basis, except for the chapters on nutrition and neoplasms.

The coverage of diseases is directly related to their importance, with good coverage of parasitic diseases, diseases of the thorax, intestinal tract, nervous system, skin, bones, endocrine system, and the reproductive system.

This is an excellent, up-to-date book for veterinarians, being the first edition of a work of its kind.—[*Diseases of Laboratory Primates. By T. C. Ruch. 1st ed. 600 pages; illustrated. W. B. Saunders Co., W. Washington Square, Philadelphia 5, Pa. 1959. Price $7.50.*]—W. C. DOLOWY.

Small Animal Proceedings

Small animal practitioners in America would be interested in the material contained in this paperbound proceedings book.—[*Proceedings of the First Annual Congress of the British Small-Animal Veterinary Association held at London, March 28-30, 1958. 136 pages; well illustrated. Published by the Association and distributed by H. K. Lewis & Co. Ltd., P.O. Box 66, 136 Gower St., London W.C. 1. Price about $6.00.*]

THE NEWS

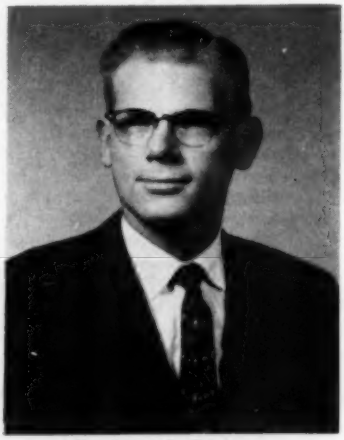
Presidents of Constituent Associations

This is the first in a series featuring the presidents of the constituent associations of the American Veterinary Medical Association. Portraits of these men will be published alphabetically according to state and province as we receive them. The AVMA salutes these veterinarians who are tirelessly working in behalf of their profession.

Arizona

Dr. Raymond E. Reed
(WSC '51)

Arkansas



Dr. James L. Forgason
(TEX '57)

Delaware

Dr. William R. Teeter
(MSC '34)

District of Columbia

Dr. Alan F. McEwan
(UP '41)

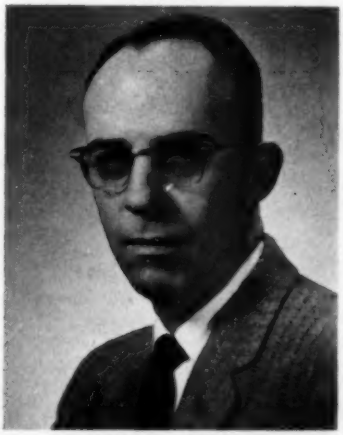
Florida

Dr. Robert P. Knowles
(API '44)

Georgia

Dr. William A. Elinburg, Jr.
(GA '51)

Indiana



Dr. L. A. Clark
(MSC '39)

Idaho

Dr. James W. Bailey
(TEX '43)

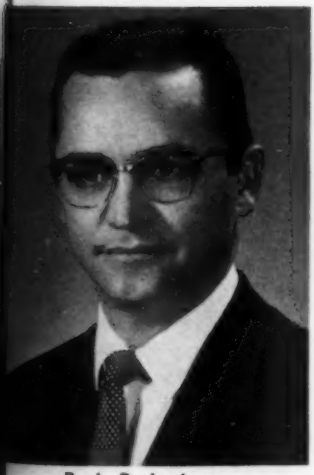
Iowa

Dr. Dale S. Adams
(ISC '39)

Kentucky

Dr. Tom S. Maddox
(API '50)

Kansas



Dr. L. D. Jernigan
(KSC '45)

Maine

Dr. Philip R. Brown
(COR '46)

Dr. W. M. Decker Elected President of the Conference of Public Health Veterinarians

Dr. W. M. Decker (MSC '46) is president of the Conference of Public Health Veterinarians for 1959. Founded in 1946, the Conference provides leadership for promoting the quality and effectiveness of veterinary public health activities which are conducted by official and nonofficial agencies and by international health organizations.

Dr. W. M. Decker

After graduation, Dr. Decker was engaged in general practice for a year and then became staff public health veterinarian in the Kalamazoo City-County Health Department for two years. From 1950 to 1955, he was milk and food consultant and then public health veterinarian for the Michigan Department of Health. He has been staff assistant to the state health commissioner since 1955.

Dr. Decker is a member of the board of directors of the Michigan V.M.A. and the Michigan Public Health Association.

s/DR. JOE W. ATKINSON, *Secretary.*

Dr. Fincher Honored for Work in Greece

Dr. Myron G. Fincher (COR '20), head, Department of Veterinary Medicine and Obstetrics, including the Ambulatory Clinic at the New York State Veterinary College, and member of the AVMA Executive Board, was made an "Honorary Professor" of the College of Veterinary Medicine at the University of Salonika, while lecturing there under the Fullbright Act.

The presentation, made Dec. 15, 1958, is a rare distinction since the only people so honored by the University in the past have been lecturers or researchers in theology, law, medicine, and philosophy. After the ceremony, Dr. Fincher delivered a lecture on "Rabies in Animals and Control in the United States." His lectures were prepared in English, translated into Greek, and presented by an interpreter. Questions and answers were also handled the same way.

Dr. Fincher accepted the invitation to lecture under the Fullbright Act from the Professional Activities Division of the International Educa-

Dr. Fincher holds a parchment scroll presented to him by Dr. Vlachos (right) at a ceremony, before a large gathering of the University and townspeople, making him an "Honorary Professor" of the University of Salonika. Some of the College's faculty and students surround them.

tional Exchange Service of the U. S. Department of State. He left the United States in July, 1958, spending a month visiting the veterinary colleges in England and on the Continent, and arrived at the University of Salonika in August.

This is the only College of Veterinary Medicine in Greece. Founded in 1950, it averages approximately 30 students to a class. The dean of the College, Dr. Konstantine Vlachos, received an M.S. degree from New York State Veterinary College in 1955.

Dr. Fincher was able to accompany clinicians into the interior of the country, visiting some villages that never had veterinary services before. In Greece, these visits are regularly scheduled so that the farmers can bring their sick animals into the villages for treatment.

Dr. Fincher and his family returned to the United States in February.

AVMA Executive Board District V Meeting

Dr. E. A. Woelffer, AVMA Executive Board member from District V (Illinois, Indiana, and Wisconsin), was chairman of the District's meeting at the LaSalle Hotel in Chicago on Feb. 15, 1959.

Attending this meeting were the three state secretaries, delegates to the AVMA House of Delegates, representatives of the state executive boards, and members of the AVMA central office staff. Topics on the agenda were: "The AVMA in Action (illustrated); The Role of AVMA Publications in Strengthening Organized Veterinary Medicine; Council Activities; Public Relation Activities;" and "Improving Communication Between Local, State, and National Associations."

Delegates to the AVMA House of Delegates who attended the Executive Board District V meeting at Chicago on February 15. Left to right—Drs. A. G. Misener, Illinois; D. E. Kelley, Wisconsin; F. R. Booth, Indiana.

State secretaries who attended the AVMA Executive Board District V meeting in Chicago on February 15. Left to right—Drs. L. M. Borst, Indiana V.M.A.; C. B. Hostetler, Illinois V.M.A.; B. A. Beach, Wisconsin V.M.A.

AMONG THE STATES AND PROVINCES

California

Death of Mrs. Wesley A. Young.—Mrs. Wesley A. (Irma) Young died on March 22, 1959, as the result of a cerebral hemorrhage.

Interment services were held at The Little Church of The Flowers at Forest Lawn Memorial Cemetery in Glendale. Surviving are her husband, one-time treasurer of the AVMA and now supervisor of the Griffith Park Zoo in Los Angeles; also a son, a daughter, and three grandchildren.

Illinois

Dr. Thiele Inspector in Chief of Meat Inspection in Chicago.—Dr. Arthur R. Thiele (KSC '34) has been advanced to inspector in charge of the Chicago, Ill., meat inspection station of the U.S.D.A. He assumed his new duties April 19, 1959. He succeeds Dr. Dale W. Glascock who was recently transferred to Omaha, Neb. (see Neb., p. 531).

Dr. Arthur R. Thiele

Dr. Thiele had formerly served as inspector in charge of the U.S.D.A.'s Newark, N.J., meat inspection station. Prior to this, he had been in New York City as assistant inspector in charge.

Dr. Thiele is a native of Bremen, Kan.

s/DR. C. H. PALS, *Associate Director.*

Iowa

Mrs. C. H. Stange Dies.—Mrs. Charles H. (Harriet Beyer) Stange, 73, widow of Dr. Stange who was dean of the Division of Veterinary Medicine at Iowa State College from 1909 to 1936 and president of the AVMA in 1923-1924, died recently in the Iowa State College Hospital.

Interment was in College Cemetery.

Kansas

Dr. Manley in Charge of Animal Disease Eradication in Kansas.—Dr. David O. Manley (KSC '41) was appointed veterinarian in charge of the U.S.D.A.'s animal disease eradication activities in Topeka, effective June 1, 1959. He exchanged assignments with Dr. Louis H. Smith, after approximately two years of similar service in Wyoming (see Wyo., p. 533).

A native of Kansas, Dr. Manley entered the U.S.D.A.'s service in that state, in 1941, in the brucellosis and tuberculosis eradication division.

He resigned in 1943 to enter private practice. In 1947, he again joined the U.S.D.A.'s service in Kansas, and was transferred to Mexico to participate in the cooperative foot-and-mouth disease eradication campaign. Dr. Manley later served in central and eastern Mexico as district supervisor with the Mexican-United States commission for the eradication of aftosa.

From 1949 until his assignment in 1956 to Cheyenne, Dr. Manley served on the field force in Kansas and as assistant veterinarian in charge in Wyoming and North Dakota.

Dr. David O. Manley

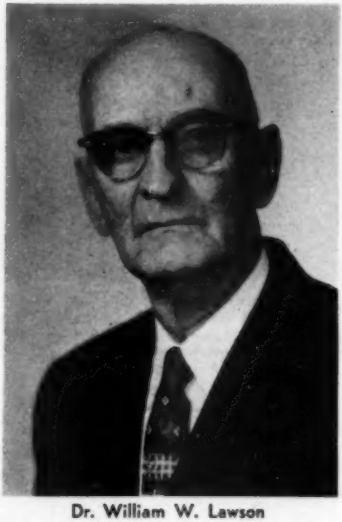
Illinois

Members of the evaluation team of the AVMA's Council on Education, are shown inspecting various records of the College of Veterinary Medicine at the University of Illinois in Urbana. They made a three-day inspection of the College's physical plant and teaching facilities Feb. 9-11, 1959.

Seated, left to right—Dr. T. L. Jones, principal, Ontario Veterinary College; Dr. J. E. Greene, dean, School of Veterinary Medicine, Alabama Polytechnic Institute; Dr. H. J. Stafseth, College of Veterinary Medicine, Michigan State. Standing—W. E. Jennings, Fifth Army Headquarters, Chicago; Dr. C. A. Brandly, dean, College of Veterinary Medicine, University of Illinois; Dr. J. R. Hay, director of AVMA professional relations.

Nebraska

Dr. Lawson Retires as Inspector in Charge of Omaha's Meat Inspection Station.—After 47 years of service in meat inspection, Dr. William W. Lawson (CVC '11) retired recently after 24 years as inspector in charge of the Omaha meat inspection station. More animals are now slaughtered in Omaha than in any city in America.



Dr. William W. Lawson

Dr. Lawson entered the meat inspection service in April, 1912, at Chicago. Throughout the years, his varied assignments took him to several locations in the U. S., including: New York City; New Orleans, La.; Allentown, Pa.; and Cincinnati, Ohio. He is a native of Oxford, Ind.

Dr. Dale W. Glascock will take Dr. Lawson's place in Omaha (see below).

s/DR. C. H. PALS, *Associate Director.*

• • •

Dr. Glascock Appointed Inspector in Charge of Meat Inspection in Omaha.—Dr. Dale W. Glascock (ISC '28) assumed the duties of inspector in charge of the Omaha meat inspection station of the U. S. D. A. on March 8, 1959. He succeeds Dr. Lawson (above).

Dr. Glascock went to Omaha, the leader in livestock slaughtering, from Chicago where he had served as inspector in charge since June, 1958. Previously, Dr. Glascock had served in the capacity of chief of the animal foods inspection section in Washington, D.C. This latter assignment had been preceded by many years of supervisory inspection assignments at various locations.

Dr. Dale W. Glascock

Dr. Glascock is a native of Audubon, Iowa.

s/DR. C. H. PALS, *Associate Director.*

New Jersey

Dr. Diehl Named Inspector in Charge of Newark's Meat Inspection.—Dr. Chester F. Diehl (UP '36) has been appointed inspector in charge of the Newark, meat inspection station

Dr. Chester F. Diehl

of the U. S. D. A., effective April 19, 1959. He succeeds Dr. A. R. Thiele (see Ill., p. 529).

Dr. Diehl went to Newark from Philadelphia, Pa., where he had served as assistant inspector in charge since 1949. Prior to this, he was stationed in Boston, Mass., and Kingston, N.Y. He is a native of Pipersville, Pa.

s/DR. C. H. PALS, *Associate Director.*

New Mexico

Bernalillo County Association.—The current roster of the Bernalillo County Veterinary Practitioners Association elected recently, includes: Drs. J. H. McCahon, president; E. L. Payne, vice-president; and Donald W. Fitzgerald, secretary-treasurer.

All the officers reside in Albuquerque.

s/DONALD W. FITZGERALD, *Secretary.*

North Carolina

Dr. Clarkson Helps Conduct Research on Atherosclerosis in Pigeons.—An article in a Winston-Salem newspaper, the *Journal and Sentinel* (Feb. 1, 1959), reported that Dr. Thomas B. Clarkson (GA '54) is currently aiding in a study on atherosclerosis in pigeons at the Bowman Gray School of Medicine, Wake Forest College, in Winston-Salem.

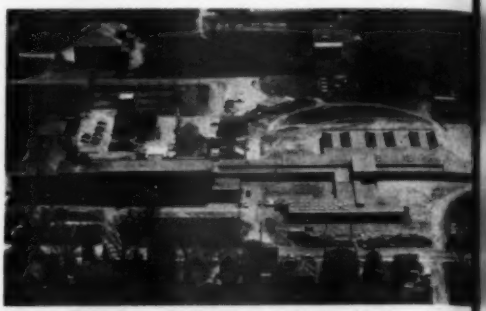
The study includes the chemistry of atherosclerosis, how it develops, how it can be prevented, and how it is affected by diet and genetics. Dr. Clarkson is chief of the laboratory in which the study is being conducted.

Pigeons are one of only two types of animals known to develop the disease naturally. The other, the baboon, is expensive and hard to procure. The 30,000 pigeons used in the research were derived from four pair of birds obtained in 1915, allowing the researchers to account for 180 pigeon generations and the equivalent of some 5,000 years of human life.

Ontario

Canadian Association.—The Canadian V.M.A. will hold its annual convention meeting at the Ontario Veterinary College in Guelph, July 20-22, 1959. One of the main features of the convention will be the official opening of the new medical surgical building on July 20.

Beside the film and television presentations, the following speakers with their respective subjects are also scheduled: Drs. W. L. Weipers, director, Veterinary Education, University of Glasgow, Scotland—veterinary education; D. G. McKercher, University of California, Davis—virus diseases in cattle; F. C. Pace, Department of National Health and Welfare, Ottawa—veterinarians in civil defense; N. L. McBride, Pasadena, Calif.—joint surgery in the dog; H. Williams, Port of Spain, Trinidad—rabies; J. N. Ritchie, Ministry of Agriculture, Surrey, England—veterinary service in the United Kingdom; D. D. Lawson, University of Glasgow, Scotland—surgery; R. Wetzel, Justus Liebig



Ontario Veterinary College in Guelph, showing the new medicine and surgery wing. The opening day ceremonies for this new building will take place during the Canadian V.M.A. meeting July 20-22.

The Canadian V.M.A.'s committee on local arrangements for the Guelph meeting. Left to right, back row—Drs. W. R. Mitchell, accommodation; A. Ferguson, exhibitors; M. C. Connell, parking; Mr. J. Busfield, secretary. Front row—Drs. J. Ballantyne, registration; C. K. Roe, entertainment; C.A.V. Barker, general chairman; J. Henderson, C.V.M.A. president; J. Archibald, C.V.M.A., vice-president and program chairman; Mrs. W. G. Stevenson, ladies and children; and Dr. J. Glover, alumni.

Veterinary College, Giessen, Germany—parasitology; J. F. Crawley, head, veterinary section, Connaught Medical Research Laboratories, Toronto—immunology; C. Thibeault, Wakefield, Mass.—horse practice; and T. J. Jones, dean, School of Veterinary Medicine, University of Georgia, Athens—nutrition.

s/DR. H. J. NEELY, *Chairman, Publicity Committee.*

Tennessee

Southern Association

Officers who attended the forty-first annual convention of the Southern V.M.A. held at the Hotel Claridge in Memphis, Oct. 26-29, 1958, are pictured above (see the JOURNAL, Jan. 1, 1959, p. 50). Left to right—E. H. Durr, Jackson, Miss., retiring president; C. C. Von Gremp, Decatur, Ga., president; M. R. Blackstock, Spartanburg, S. Car., treasurer; A. A. Husman, Raleigh, N. Car., secretary.

Texas

El Paso V.M.A. Helps Sponsor Livestock Forum.—El Paso's first Livestock Forum held in January, 1959, was sponsored jointly by the El Paso V.M.A. and *The El Paso Times,* a local newspaper. Representatives from west Texas and southern New Mexico attended the meeting held in the El Paso Electric Auditorium.

Speakers outlined the problems of the livestock industry and the research being done to overcome them. A series of small animal forums were tentatively planned due to the success of this first venture.

Wyoming

Dr. Smith in Charge of Animal Disease Eradication Activities in Wyoming.—Dr. Louis H. Smith (KSC '28) was named veterinarian in charge of the U.S.D.A.'s animal disease eradication activities in Cheyenne, effective June 1, 1959. He had been stationed in Topeka, Kan., as veterinarian in charge of animal disease eradication activities, since May, 1955. He is exchanging locations with Dr. David Manley who was veterinarian in charge in Wyoming before his transfer (see Kan., p. 530).

After graduation, Dr. Smith was engaged in private practice in his home state of Kansas until 1934 when he joined the BAI. In 1935, he went to Texas, returning to field service on brucellosis and tuberculosis work in Kansas in 1936. In 1947, he was transferred to Mexico to participate in the campaign against foot-and-mouth disease (aftosa) as district supervisor.

Dr. Louis H. Smith

Dr. Smith then returned to the Kansas field force for a year. In 1949, he was sent to Indiana on a BAI and Public Health Service project, correlating animal and human brucellosis, until his transfer to Kansas in 1955.

FOREIGN NEWS

Australia

General Meeting.—The thirty-sixth annual meeting of the Australian Veterinary Association was held at the University of Queensland, St. Lucia, Brisbane, May 25-29, 1959.

Dr. Louis C. Heemstra (ISC '32), Beltsville, Md., represented the AVMA at the conference.

STATE BOARD EXAMINATIONS

FLORIDA—June 15-17, 1959, Biscayne Terrace Hotel, Miami. Address all inquiries to Dr. E. L. Matthews, Secretary of the Board of Veterinary Examiners, Box 141, Palatka, Fla. Deadline for acceptance of applications will be June 1, 1959.

IOWA—June 1-2, 1959, Office of the Division of Animals Industry, State House, Des Moines, Iowa, not later than 8 o'clock on the morning of June 1. Further information may be obtained from Dr. A. L. Sundberg, Chief, Division of Animal Industry, State House, Des Moines 19, Iowa.

KENTUCKY—June 17, 1959, State Capitol Building, Frankfort, Ky. Jack E. Winkler, Secretary-Treasurer, Kentucky Board of Veterinary Examiners, 319 Ann St., Frankfort, Ky.

MARYLAND—June 8-9, 1959, College Park, at 8:00 a.m. Applications must be returned to the secretary, no later

than June 1. Dr. Harold S. Gober, 5400 Park Heights Ave., Baltimore 15, Md., secretary-treasurer.

MASSACHUSETTS—June 25-27, 1959, University of Massachusetts, Amherst. Address inquiries to Dr. Edward A. Blake, Secretary, Board of Registration in Veterinary Medicine, Room 33, State House, Boston 33, Mass.

MICHIGAN—June 12-13, 1959, Lansing, Mich. These will be written, practical, and oral examinations. Applications must be on file at least 15 days before the exam, accompanied by the $25 fee. Address: State Veterinarian, 641 Lewis Cass Building, Lansing 13, Mich., John F. Quinn, corresponding secretary.

MISSOURI—June 5-6, 1959, Veterinary Clinic, University of Missouri, Columbia. Application blanks may be obtained from Dr. L. A. Rosner, Board Chairman, Box 630, Jefferson City, Mo.

MONTANA—June 22-24, 1959, Montana Veterinary Research Laboratory, Montana State College, Bozeman, Mont. For application blanks, write: Dr. J. W. Safford, Secretary-Treasurer, State Board of Veterinary Medical Examiners, Capitol Station, Helena, Mont.

NORTH CAROLINA—June 22-24, 1959, Morehead Biltmore Hotel, Morehead City, N. Car. Dr. James I. Cornwell, Secretary-Treasurer, North Carolina State Veterinary Examining Board, 65 Beverly Rd., Beverly Hills, Asheville, N. Car.

OHIO—June 8-10, 1959, Sisson Hall, College of Veterinary Medicine, Ohio State University, Columbus, Ohio. Applicants must be present at 8 a. m. on the first day. Application forms may be obtained from the office of the Executive Secretary, Ohio Veterinary Medical Board, Room 720, Ohio Departments Building, Columbus 15. All application forms must be returned to the Secretary not later than May 4, 1959. Dr. H. G. Geyer, executive secretary.

OREGON—June 15-17, 1959, Hotel Multnomah, Portland. Dr. C. R. Howarth, Secretary, Oregon State Veterinary Medical Examining Board, 135 N. Highway, St. Helens, Ore.

SOUTH DAKOTA—June 22-23, 1959, Brookings. Applications should be sent to Office of Livestock Sanitary Board, Pierre, S. Dak., Dr. M. D. Mitchell, state veterinarian.

TEXAS—Next licensing examination will be held June 1-3, 1959, A. & M. College of Texas, College Station. The completed application must be received in the Board office not later than 30 days before the examination date. Applications should be sent to Mr. T. D. Weaver, 207 Capital National Bank Building, Austin 16, executive secretary, State Board of Veterinary Medical Examiners.

UTAH—June 11-12, 1959, Utah State Capitol Building, Salt Lake City, Utah. Dr. Wayne Binns, Chairman, Utah Veterinary Examining Board, 555 North Third East, Logan, Utah.

WEST VIRGINIA—June 15, 1959, Capitol Building, Room 117, Charleston. For information and application forms, write: Dr. Harry J. Fallon, 200 Fifth St. West, Huntington, W. Va., secretary-treasurer.

WISCONSIN—June 29-30, 1959, Madison, Wis. A. A. Erdmann, 6 West St., State Capitol, Madison 2, Wis.

WYOMING—June 18, 19, 1959, State Capitol, Cheyenne. For further details write Dr. G. H. Good, 304 Capitol Bldg., Cheyenne, Wyo., secretary.

DEATHS

Star indicates member of AVMA

John W. Foley (ONT '17), 63, Endeavor, Wis., died Feb. 28, 1959, from a heart attack.

★**Sidney Nathanson** (COR '36), 48, East Rockaway, L.I., N.Y., died from a coronary occlusion on Aug. 18, 1958.

Associated in practice with Drs. Barrett and Noonan of Akron, Ohio, for a short interval after graduation, Dr. Nathanson then returned to New York where he began his own practice. During World War II, he served as a major in the Army Veterinary Corps and later was discharged as a lieutenant colonel.

Dr. Nathanson was chairman of the ethics committee of the New York City V. M. A. for several years and a member of the Metropolitan Veterinary Practitioners Club.

Morris W. Ray (KCV '11), 74, Longview, Wash., died in an automobile accident near Longview on Jan. 4, 1959. He had retired in 1944 due to ill health.

After graduation, Dr. Ray practiced in Clark, S. Dak., for 24 years.

In 1925, he was appointed state veterinarian in South Dakota, and in 1929, he joined Corn States Serum Company where he remained until his retirement.

Dr. Ray was president of the South Dakota V. M. A. from 1923 to 1925.

William C. Vollstedt (CVC '11), 70, Dixon Iowa, died in Mercy Hospital there on March 31, 1959. He had been in ill health for a week.

Born in Davenport, Dr. Vollstedt practiced there until 1925 when he moved to Dixon where he once served as mayor.

• • •

Other Deaths Reported.—The following deaths have been reported. The usual information for an obituary was not supplied.

Ralph W. Boyce (HAR '01), 81, Sebring, Fla., died Feb. 12, 1959.

Harry C. Evers (ISC '10), 73, Denver, Colo., died Feb. 3, 1959.

L. R. Fauteck (KCV '06), 76, Wichita, Kan., died Jan. 26, 1959.

George H. Hines (IND '21), 60, Monticello, Ill., died Jan. 16, 1959.

V. J. Laurent (MCK '16), 76, Luxemburg, Wis., died Jan. 29, 1959.

Ray O. Porter (KCV '09), 79, San Luis Obispo, Calif., died Feb. 7, 1959.

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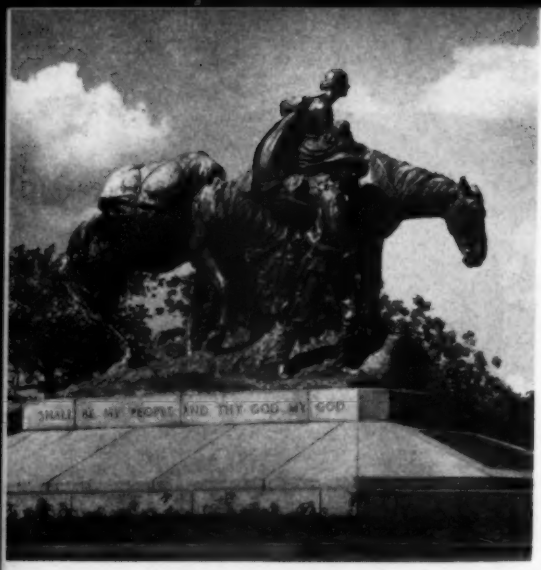


"Pioneer Mother"—a sculptural group located in Penn Valley Park, Kansas City. It is a tribute to the courage of the women who braved hardship and danger in the settlement of the West.

Joint AVMA-Pan American Meeting

Kansas City, August 24-27

Missouri — Gateway to the West

Missouri is rich in the history of the development of the West. In 1541, Hernando DeSoto of Spain was the first white man to see the rolling hills and wooded banks of the Missouri lakes and rivers. In 1682, LaSalle took possession in the name of France. The state's territory was added to the United States by the Louisiana Purchase of 1803.

Lewis and Clark set out from St. Louis, Mo., in 1804 to explore the Pacific Northwest, using the broad waters of the Missouri as an avenue to the West. Enroute they passed Boonville (now Franklin, Mo.), the last settlement established by Daniel Boone.

St. Louis, at the junction of the Mississippi and Missouri Rivers, was the natural jumping-off point for parties heading west overland. The mountain men poled and kedged up the Missouri to the junction with the Kaw River, fighting and trading with the Indians for beaver pelts.

Franklin was the initial starting point of the Santa Fe Trail. As the pressure of civilization pushed from the East, the head of the trail moved west to Independence. The Oregon and California Overland Trails started at Westport Landing (now Kansas City).

In 1860, the Pony Express linked St. Joseph, Mo., and points east to Sacramento, Calif., and the West.

Organized as a state in 1812, the slavery issue delayed Missouri's statehood until 1821. The Missouri Compromise made it the 24th state, and the first west of the Mississippi. During the Civil War, Missouri furnished troops to both sides, and though nominally a Union state, her fields and woods, towns and villages, were the scenes of bloody guerilla battles.

Her history has left marks on the landscape. Zigzag wood fences, a weathered gray log cabin, a rustic mill, a covered bridge, are integral parts of the past surviving into the present. Other

Arrow Rock Tavern, on the old Santa Fe Trail. Independence and Kansas City served as the last outposts of civilization for the settlers in wagon trains, who stocked up on provisions and equipment, and prepared for the long and painful journey westward across the plains.

Photos courtesy of Missouri Resources Division.

historic events are memorialized in monuments and markers, providing an education for the youngsters in the history of America's westward expansion.

For explorers, Missouri has 21 caverns throughout the state which are easily accessible from main highways. Some are large enough to accommodate thousands of persons. One which has been immortalized in story is Mark Twain Cavern, two miles south of Hannibal, Mo., on U. S. 36-61. This cave was probably the inspiration for the adventures of Tom Sawyer and Becky Thatcher.

Attractions near Kansas City include Fort Osage at Buckner, Mo., the Confederate Memorial at Higginsville; and the Battlefield and Old Court House at Lexington. Places of interest in Kansas City are the Pioneer Mothers Memorial, Liberty Memorial, Starlight Theatre, Swope Park, Nelson Art Gallery, Kansas City University, Rockhurst College, and the Kansas City Athletics baseball park.

Spanish Conversation

Habla Español?

Since the AVMA Convention in Kansas City will be held jointly with the Pan-American Congress, with approximately 400 Spanish-speaking veterinarians and visitors, the next four issues of the JOURNAL will carry phrases of simplified Spanish conversation.

Although conscientious study of these little lessons will not transform you into a fluent linguist, you may find that to your satisfaction, you can exchange greetings with our Latin American neighbors in Kansas City.

Como Esta Usted? *KO-mo eh-STAH oo-STETH?*	How are you?
Muy bien, gracias *MWEE be-AYN GRATH-ias*	Fine, thank you
Y Usted? *ee oo-STETH?*	And you?
Buenos dias *BWAY-nos DEE-ahs*	How do you do Good morning
Buenos tardes *BWAY-nos TAR-days*	Good afternoon
Buenos noches *BWAY-nos NO-chez*	Good evening
Quiero presentarle al senor . . . *kee-AIR-o pre-zen-TAHR-ley al seyn-YOR . . .*	May I introduce you to . . .
Tengo tanto gusto en haberle conocido *TENG-go TAHN-toe GOOS-toe ayn ah-BARE-ley ko-no-SEE-do*	I am very glad to have met you
Adios *ah-dee-OS*	Good-bye

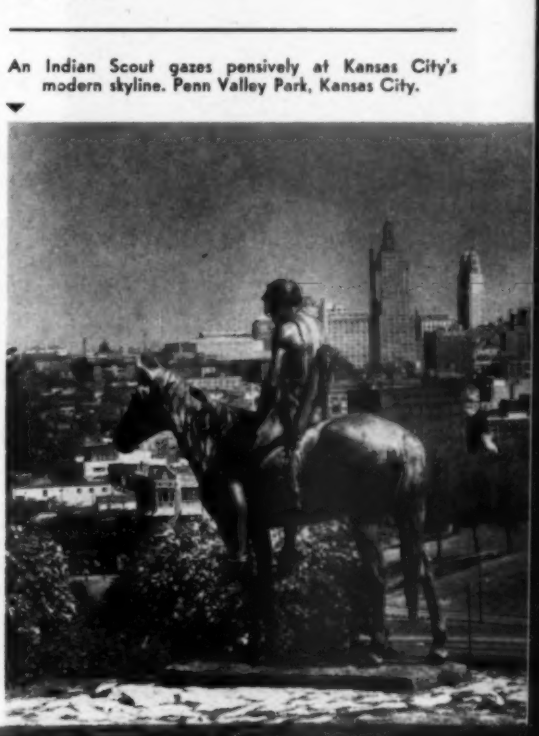
Fort Osage, built in 1808, was a fortified outpost on the U. S. frontier. Only 20 miles from downtown Kansas City, it is situated on a point overlooking the Missouri River.

Kansas City Housing

Hotel information, rates, and reservation form will be found on adv. pages 45 and 46 of this issue, facing a location map for hotels and some other centrally located points of interest on adv. page 45.

The Kansas City Convention and Visitors Bureau will handle reservations. Early reservations are advised.

An Indian Scout gazes pensively at Kansas City's modern skyline. Penn Valley Park, Kansas City.



Proposed Amendments to Constitution and Bylaws

The following amendments are published in accordance with Article XI of the Bylaws and will, therefore, be in order for final action at the 1959 session of the House of Delegates.

New Proposals

AMENDMENT No. 1

Amend Article I, Section 1, Bylaws, to read:

Active members.—(*a*) A member in good standing of a constituent association (as certified by the secretary thereof to the executive secretary) shall become an active member of the Association if he meets the requirements set forth in Article III of the Constitution and is approved for membership by the Judicial Council. *An active member must continue to retain membership in a constituent association in order to remain an active member in good standing in this Association.*

AMENDMENT No. 2

Amend Article II, Section 9 (c), Bylaws, to read:

c) Pay all expenses of the Association, including drafts for the revolving fund, subject to the direction and disposition of the Executive Board.

AMENDMENT No. 3

Amend Article VIII, Section 2, Bylaws, to read:

Section 2—Location.—For the purpose of determining where the annual session is to be held, the United States and the Dominion of Canada shall be divided into *five* convention zones as shown on the official map herewith reproduced. The zone in which the annual session is to be held shall be as follows:

Zone 1—Every fifth year beginning with the year 1964.

Zone 2—Every fifth year beginning with the year 1961.

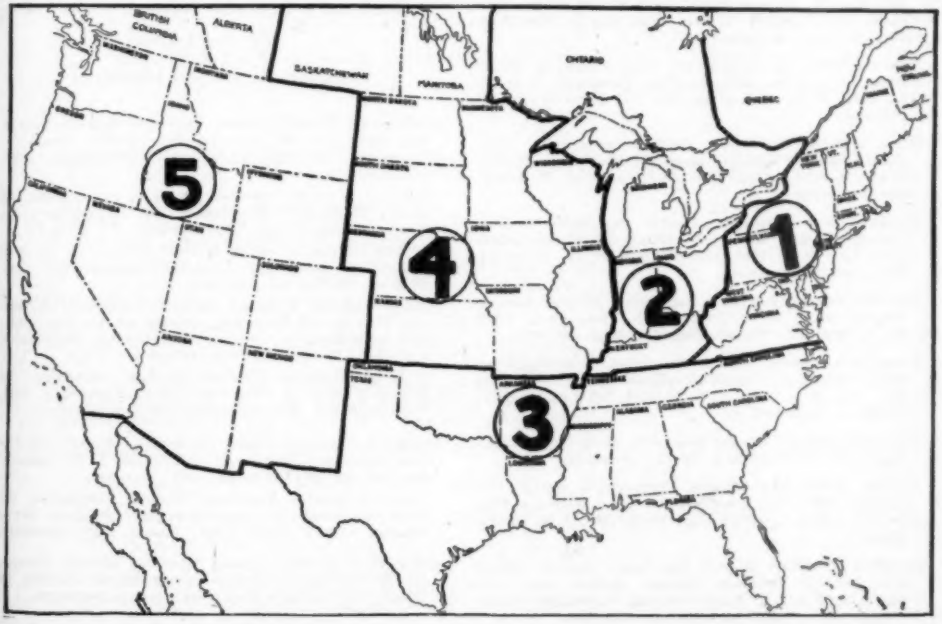
Zone 3—Every fifth year beginning with the year 1962.

Zone 4—Every fifth year beginning with the year 1963.

Zone 5—Every fifth year beginning with the year 1960.

The city within the convention zone in which the annual session for a calendar year is to be held shall be determined by the House of Delegates at its annual meeting held not less than two years preceding such calendar year.

Apportionment of Areas for AVMA Conventions



COMING MEETINGS

Notices of coming meetings must be received 30 days before date of publication.

Texas, A. & M. College of. Twelfth annual conference. School of Veterinary Medicine, A. & M. College of Texas, College Station, June 4-5, 1959. R. D. Turk, chairman.

Tulsa Veterinary Medical Association. Summer meeting. Western Hills Lodge, Box 276, Wagoner, Okla., June 14-15, 1959. Arlen D. Hill, 5302 E. 11th St., Tulsa, Okla., secretary.

Georgia Veterinary Medical Association. Annual meeting. Jekyll Hotel, Jekyll Island, Ga., June 15-16, 1959. A M. Mills, 325 Pinecrest Dr., Athens, Ga., secretary.

Ohio State University. Annual spring conference. College of Veterinary Medicine, Ohio State University, Columbus, June 16-17, 1959.

Utah Veterinary Medical Association. Annual meeting. Provo, June 17-18, 1959. J. A. Thomas, P.O. Box 592, Provo, Utah, secretary-treasurer.

Illinois, University of. Fourth Biennial Symposium on Animal Reproduction. University of Illinois, Urbana, June 18-20, 1959. Address P. J. Dziuk, 111 Animal Genetics, University of Illinois, Urbana, for further information.

South Carolina Association of Veterinarians. Annual summer meeting. Clemson House, Clemson, S. Car., June 18-20, 1959. B. C. McLean, 808 Linden St., Aiken, S. Car., program chairman.

Alberta Veterinary Medical Association. Annual convention. Jubilee Auditorium, Edmonton, June 19-20, 1959. H. N. Vance, 14323-106 A Ave., Edmonton, Alta., chairman.

Louisiana Veterinary Medical Association, Inc. Annual meeting. Virginia Hotel, Monroe, La., June 21-23, 1959. Dr. Walter Ernst, Jr., P.O. Box 2502, Monroe, La., local arrangements committee chairman.

California Veterinary Medical Association. Seventy-first annual meeting. Miramar Hotel, Santa Monica, Calif., June 21-23, 1959. Chas. S. Travers, 3004 16th St., San Francisco, executive secretary.

Maritime Veterinary Associations, joint conference. Mount Allison University, Sackville, New Brunswick, June 23-25, 1959. P. D. McKercher, Box 310, Sackville, general chairman.

North Carolina Veterinary Medical Association. Fifty-eighth annual summer meeting. Morehead Biltmore Hotel, Morehead City, June 23-25, 1959. J. T. Dixon, secretary-treasurer.

Maryland State Veterinary Medical Association. Summer meeting. George Washington Hotel, Ocean City, Maryland, June 25-26, 1959. Harry L. Schultz, Jr., Baltimore, Md., secretary-treasurer.

Nebraska Veterinary Medical Association. Summer meeting. Pawnee Hotel, North Platte, Neb., July 10-12, 1959. H. E. Hedlund, Wahoo, Neb., program chairman.

Kentucky Veterinary Medical Association. Forty-eighth annual convention. Sheraton-Seelbach Hotel, Louisville, Ky., July 13-14, 1959. L. S. Shirrell, 545 East Main St., Frankfort, secretary.

Iowa State College. Annual conference. Iowa State College, Ames, July 14-15, 1959. C. D. Lee, chairman.

Alabama Polytechnic Institute. Fifty-second annual conference. School of Veterinary Medicine, Alabama Polytechnic Institute, Auburn, July 19-22, 1959. J. E. Greene, dean.

Canadian Veterinary Medical Association. Annual convention. Ontario Veterinary College, Guelph, Ont., July 20-22, 1959. H. J. Neely, Ontario Veterinary College, Guelph, Ont., publicity committee chairman.

American Association of Veterinary Bacteriologists. Annual meeting. Division of Veterinary Medicine. Iowa State College, Ames, Iowa, Aug. 22, 1959. C. H. Cunningham, Michigan State University, College of Veterinary Medicine, East Lansing, secretary.

Ninety-Sixth Annual Meeting, American Veterinary Medical Association, and Third Pan American Congress of Veterinary Medicine. Joint meeting. Kansas City, Mo., Aug. 23-27, 1959. H. E. Kingman, Jr., executive-secretary, AVMA, 600 S. Michigan Ave., Chicago 5, Ill. B. D. Blood, secretary-general, Directing Council, Pan American Congress of Veterinary Medicine, P.O. Box 99, Azul, F.C.N.G.R., Argentina, S.A.

New Mexico Veterinary Medical Association. Annual meeting, Western Skies Hotel, Albuquerque, N. M., Sept. 21-22, 1959. E. R. Leslie, 907 Alamosa, Carlsbad, N.M., secretary.

Foreign Meetings

Veterinary Symposium in Israel (with International Farmers Convention); Veterinary Institute, Beit-Dagan, Israel, May 10-12, 1959. Dr. A. Kimron, Veterinary Institute, director. (Particulars are also obtainable from Dr. H. E. Newman, American Veterinarians for Israel, Box 145, Merrifield, Va.

International Veterinary Congress. Sixteenth session. Madrid, Spain, May 21-27, 1959. Prof. Pedro Carda A., general secretary, Calle Villanueva 11, Madrid.

U.S. COMMITTEE: Dr. W. A. Hagan, chairman, New York State Veterinary College, Ithaca, N. Y.; Dr. J. G. Hardenbergh, secretary, 600 S. Michigan Ave., Chicago 5, Ill.

Third World Congress on Fertility and Sterility. Amsterdam, Holland, June 7-13, 1959. Dr. L. I. Swaab, Sint Agnietenstraat 4, Amsterdam, Holland, honorary secretary.

Regularly Scheduled Meetings

ALABAMA—Central Alabama Veterinary Medical Association, the first Thursday of each month. James L. Chambers, 4307 Normanbridge Rd., Montgomery, Ala., secretary-treasurer.

Jefferson County Veterinary Medical Association, the second Thursday of each month. Dan P. Griswold, Jr., 714 S. 39th St., Birmingham, secretary.

Mobile-Baldwin Veterinary Medical Association, the third Tuesday of each month. Cecil S. Yarbrough, 4121 U.S. 90 West, Mobile, Ala., secretary.

North Alabama Veterinary Medical Association, the second Thursday of November, January, March, May, July, and September, in Decatur, Ala. Ray A. Ashwander, P.O. Box 1767, Decatur, Ala., secretary.

Northeast Alabama Veterinary Medical Association, the second Tuesday of every other month. Leonard J. Hill, P.O. Box 761, Gadsden, Ala., secretary-treasurer.

ARIZONA—Central Arizona Veterinary Medical Association, the second Tuesday of each month. J. W. Langley, Jr., P.O. Box 5013, Phoenix, Ariz., secretary.

Southern Arizona Veterinary Medical Association, the third Wednesday of each month at 7:30 p.m. Gwyn Chapin, 2215 E. Calle Vista, Tuscon, Ariz., secretary.

ARKANSAS—Pulaski County Veterinary Medical Society, the second Tuesday of each month. Harvie R. Ellis, 54 Belmont Drive, Little Rock, Ark., secretary-treasurer.

(Continued on adv. p. 34)

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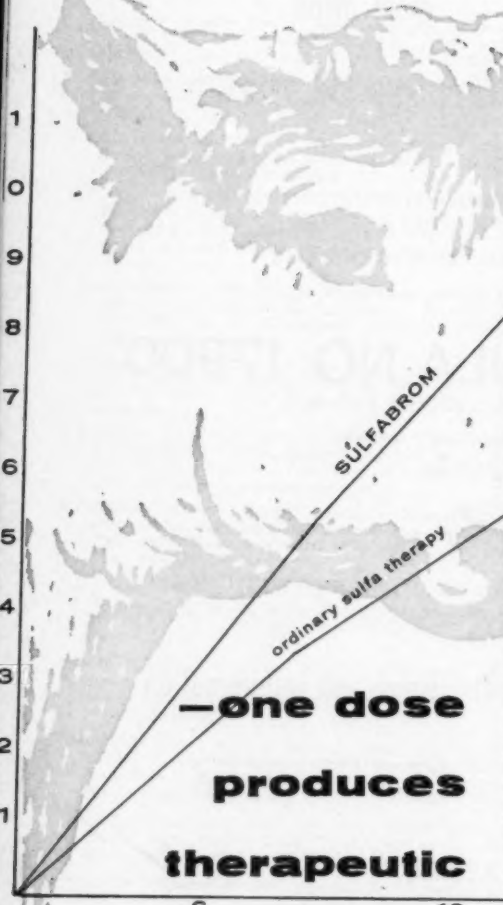

—one dose produces therapeutic blood levels lasting up to 48 hours in cattle

More than any other professional man, the practicing veterinarian knows that his most valuable asset is time. In any area of his practice, the success of his therapy cannot be measured by results alone; he is a busy man whose time must be used judiciously.

Well aware of this problem, Merck research workers have designed an antibacterial agent which, from its conception, was meant to satisfy the particular needs of the veterinarian. In cattle, this new drug produces effective therapeutic blood levels for at least two days, freeing the veterinarian from time-consuming return calls and minimizing stress-producing handling.

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intraperitoneally to cattle, produces effective blood levels lasting up to 48 hours—frequently long enough to eliminate *any* repetition of dosage.

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Although SULFABROM is notable for producing effective levels in rapid time, once it has entered the blood stream its speed of action slows down considerably. SULFABROM is excreted very slowly; this accounts for its long-lasting effect. In cattle, detectable amounts may be present in the urine for as long as six days. Blood levels remain high, sometimes for as long as 53 to 60 hours. And, because it is excreted so slowly, seldom is the amount of SULFABROM passing through the urinary tract ever large enough to cause crystalluria.

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foot rot "in 48 hours the cow was able to stand and started to eat";
pneumonia "in five days' time this calf was back to normal";[1]
as well as *scours*, winter dysentery, coccidiosis, shipping fever, listerellosis and miscellaneous infectious conditions, such as peritonitis and infected wounds.

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15-30 mg. (0.25-0.5 grains)/lb., intravenously—for *immediate* levels
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SULFABROM 4 Gm. Boluses—packages of 5 and 50

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VETERINARY

1. CASE REPORTS IN MERCK & CO., INC. FILES.

CALIFORNIA—Alameda-Contra Costa Veterinary Medical Association, the fourth Wednesday of Jan., March, May, June, Aug., Oct., and Nov. John S. Blackard, 420 Appian Way, Richmond, Calif., secretary.

Bay Counties Veterinary Medical Association, the second Tuesday of February, April, July, September, and December. Herb Warren, 3004 16th St., San Francisco, Calif., executive secretary.

Central California Veterinary Medical Association, the fourth Tuesday of each month. Paul S. Chaffee, 2333 McKinley Ave., Fresno, Calif., secretary.

Humboldt-Del Norte Counties Veterinary Medical Association, the second Tuesday of January, May, September, and November. Dr. C. A. Lamb, 2835 Dolbeer St., Eureka, Calif., secretary.

Kern County Veterinary Medical Association, the first Thursday evening of the month. James L. Frederickson, 17 Nile St., Bakersfield, Calif., secretary-treasurer.

Mid-Coast Veterinary Medical Association, the first Thursday of each month. William P. Matulich, P. O. Box 121, San Luis Obispo, Calif., secretary-treasurer.

Monterey Bay Area Veterinary Medical Association, the third Wednesday of each month. V. Todorovic, 47 Mann Ave., Watsonville, Calif., secretary.

Northern California Association of Veterinarians, the second Tuesday of the month. Andrew F. Giambroni, P.O. Box 782, Red Bluff, Calif., secretary.

North San Joaquin Valley Veterinary Medical Association, the fourth Wednesday of each month at the Hotel Covell, in Modesto, Calif. T. J. Carleton, 325 W. Lockeford St., Lodi, Calif., secretary-treasurer.

Orange Belt Veterinary Medical Association, the second Monday of each month. R. Y. Foos, P.O. Box 955, Victorville, Calif., secretary-treasurer.

Orange County Veterinary Medical Association, the third Thursday of each month. H. M. Stanton, 1122 S.E. U.S. Highway 101, Tustin, Calif., secretary.

Peninsula Veterinary Medical Association, the third Monday of the month. R. M. Grandfield, 416 Stephens Rd., San Mateo, Calif., secretary-treasurer.

Redwood Empire Veterinary Medical Association, the third Thursday of the month. R. R. Rediske, 833 Vallejo Ave., Novato, Calif., secretary-treasurer.

Sacramento Valley Veterinary Medical Association, the second Wednesday of the month. E. C. Story, 4819 "V" St., Sacramento 17, Calif., secretary-treasurer.

San Diego County Veterinary Medical Association, the fourth Tuesday of the month. Robert F. Burns, 7572 North Ave., Lemon Grove, Calif., secretary-treasurer.

San Fernando Valley Chapter SCVMA, the second Tuesday of each month at 7:30 p.m., Hody's Restaurant, North Hollywood, Calif. Dr. V. H. Austin, 14931 Oxnard St., Van Nuys, secretary-treasurer.

San Fernando Valley Veterinary Medical Association, the second Friday of each month at the Casa Escobar Restaurant in Studio City. John Chudacoff, 7912 Sepulveda Blvd., Van Nuys, Calif., secretary.

Santa Barbara-Ventura Counties Veterinary Medical Association, every three months, no set date. Gerald M. Clark, 5415 8th St., Carpinteria, Calif., secretary-treasurer.

Santa Clara Valley Veterinary Medical Association, the last Tuesday of the month. Robert L. King, 1269 Grant St., Santa Clara, Calif., secretary-treasurer.

Southern California Veterinary Medical Association, the third Wednesday of the month. Mr. Don Mahan, 1919 Wilshire Blvd., Los Angeles 57, Calif., executive secretary.

COLORADO—Denver Area Veterinary Medical Society, the fourth Tuesday of every month. Gene M. Bierhaus,

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REFERENCES: 1. Mosier, J. E., and Coles, E. H.: Vet. Med. 53:649 (Dec.) 1958. 2. Belloff, G. B.: Calif. Vet. 9:27 (Sept.-Oct.) 1956. 3. Mosier, J. E.: Vet. Med. 52:445 (Sept.) 1957.

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EATON LABORATORIES, NORWICH, NEW YORK

2896 S. Federal Blvd., Englewood, Colo., secretary-treasurer.

Northern Colorado Veterinary Medical Society, the first Wednesday of each month, in Fort Collins. Dr. James Voss, Veterinary Hospital, Colorado State University, Fort Collins, Colo., secretary.

DELAWARE—New Castle County Veterinary Medical Association, the first Tuesday of each month at 9:00 p.m. in the Hotel Rodney, Wilmington, Del. A. P. Mayer, Jr., R.F.D. 2, Newark, Del., secretary-treasurer.

DISTRICT OF COLUMBIA—District of Columbia Veterinary Medical Association, the second Tuesday evenings of January, March, May, and October. R. B. Gochenour, 10109 Ashwood Dr., Kensington, Md., secretary-treasurer.

FLORIDA—Big Bend Veterinary Medical Association, meets the first Sunday of each month at 5:00 p. m., at the Tallahassee Dining Room, Tallahassee. C. Paul Vickers, P.O. Box 309, Tallahassee, secretary.

Central Florida Veterinary Medical Association, the first Friday of each month at 8:00 p. m., place specified monthly. L. R. Poe, 753 W. Fairbanks Ave., Winter Park, Fla., secretary-treasurer.

Florida West Coast Veterinary Medical Association, the second Wednesday of each month at the Lighthouse Inn, St. Petersburg. Fred Jones, 3606 S. Dale Mabry, Tampa, Fla., secretary.

Jacksonville Veterinary Medical Association, the first Thursday of every month. Dodson's Restaurant, Stephen C. Hite, 5807 105th St., Jacksonville 10, Fla., secretary.

Northwest Florida Veterinary Medical Society, third Wednesday of each month, time and place specified monthly. John Webb, P.O. Box 183, Cantonment, Fla., secretary-treasurer.

Palm Beach Veterinary Society, the last Thursday evening of each month. McArthur Dairy Building, Four Points, W. Palm Beach. B. W. Bigger, 2833 S. 4th St., Fort Pierce, Fla., secretary.

Ridge Veterinary Medical Association, the fourth Thursday of each month in Bartow, Fla. John S. Haromy, Route #1, Box 107-A, Lake Wales, Fla., secretary.

South Florida Veterinary Society, the third Wednesday of each month. Time and place specified monthly. Joe B. O'Quinn, 1690 E. 4th, Hialeah, Fla., secretary.

Suwannee Valley Veterinary Association, the fourth Tuesday of each month, Hotel Thomas, Gainesville. G. L. Burch, P.O. Box 405, Ocala, Fla., secretary-treasurer.

Volusia County Veterinary Medical Association, the fourth Thursday of each month. Robert E. Cope, 127 E. Mason, Daytona Beach, Fla., secretary.

GEORGIA—Atlanta Veterinary Medical Society, the third Thursday of each month at the Elk's Home, 726 Peachtree St., Atlanta. Clare L. Bromley, 634 Northside Dr., N.W., Atlanta, Ga., secretary.

Georgia-Carolina Veterinary Medical Association, the second Monday of each month at 8:00 p.m., at the Town Tavern, Augusta, Ga. J. A. Schmitz, 1711 Gwinnett St., Augusta, Ga., secretary.

North Georgia Veterinary Medical Association, quarterly, no set date, the spring meeting at the Veterinary School, Athens, Ga. S. J. Shirley, Commerce, Ga., secretary.

Southeast Georgia Veterinary Medical Association, quarterly, date and meeting place varies. Hugh F. Arundel, P.O. Box 153, Statesboro, Ga., secretary.

South Georgia Veterinary Medical Association, the second Sunday of each quarter at 3:30 p.m., at the Radium Springs Hotel, Albany, Ga. M. W. Hale, Route 2, Tifton, Ga., secretary.

ILLINOIS—Central Illinois Veterinary Medical Association,

June 9, Sept. 9, and Dec. 10, 1959. Paul B. Doby, 4 Owens Lane, Springfield, secretary.

Chicago Veterinary Medical Association, the second Tuesday of each month, Charles H. Armstrong, 1021 Davis St., Evanston, secretary.

INDIANA—Central Indiana Veterinary Medical Association, the second Wednesday of each month. P. T. Parker, 224 N. Mill St., secretary-treasurer.

Michiana Veterinary Medical Association, the second Thursday of every month except July and December, at the Hotel LaSalle, South Bend, Ind. Stanton Williamson, 217 W. Chippewa St., South Bend, Ind., secretary.

Tenth District Veterinary Medical Association, the third Thursday of each month. J. S. Baker, P.O. Box 52, Pendleton, Ind., secretary.

IOWA—Cedar Valley Veterinary Medical Association, the second Monday of each month, except January, July, August, and October in Black's Tea Room, Waterloo, Iowa. A. J. Cotten, P.O. Box 183, Grundy Center, secretary.

Central Iowa Veterinary Medical Association, the third Monday of each month except June, July, and August at 6:30 p.m., Breeze House, Ankeny, Iowa. S. L. Hendricks, secretary-treasurer .

Central Iowa Veterinary Medical Association, the third Monday of each month, except June, July, and August, at 6:30 p.m., Breeze House, Ankeny, Iowa. John Herrick, 202 S. Hazel Ave., Ames, secretary.

Coon Valley Veterinary Medical Association, the second Wednesday of each month, September through May, at 7:30 p.m., Cobblestone Inn, Storm Lake, Iowa. Robert McCutcheon, Holstein, secretary.

East Central Iowa Veterinary Medical Society, the Second Thursday of each month at 6:30 p.m., usually in Cedar Rapids, Iowa. T. F. Bartley, P.O. Box 454, Cedar Rapids, secretary.

Fayette County Veterinary Medical Association, the third Thursday of each month at 6:30 p.m. in West Union, Iowa. H. J. Morgan, West Union, secretary.

Lakes Veterinary Association, the first Tuesday of each

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month, September through May, at 6:30 p.m., at the Gardson Hotel, Estherville, Iowa. Barry Barnes, P.O. Box 162, Milford, secretary.

North Central Iowa Veterinary Medical Association, the third Thursday of April, at the Warden Hotel, Fort Dodge, Iowa. H. Engelbrecht, P. O. Box 797, Fort Dodge, secretary.

Northeast Iowa-Southern Minnesota Veterinary Association, the first Tuesday of February, May, August, and November at the Wisneslick Hotel, Decorah, Iowa, 6:30 p.m. Donald E. Moore, Box 178, Decorah, Iowa, secretary.

Northwest Iowa Veterinary Medical Association, the second Tuesday of February, May, September, and December, at the Community Bldg., Sheldon. W. Ver Meer Hull, secretary.

Southeastern Iowa Veterinary Association, the first Tuesday of each month at Mt. Pleasant, Iowa. Warren Kilpatrick, Mediapolis, secretary.

Southwestern Iowa Veterinary Medical Association, the first Tuesday of April and October, Hotel Chieftain, Council Bluffs, Iowa. J. P. Stream, 202 S. Stone St., Creston, secretary.

Upper Iowa Veterinary Medical Association, the third Tuesday of each month at 7:00 p.m., at All Vets Center, Clear Lake, Iowa. W. A. Danker, Dows, Iowa, secretary.

KENTUCKY—Central Kentucky Veterinary Medical Association, the first Wednesday of each month. R. H. Folsom, P.O. Box 323, Danville, Ky., secretary.

Jefferson County Veterinary Society of Kentucky, Inc., the first Wednesday of each month in Louisville or within a radius of 50 miles, except January, May, and July. G. R. Comfort, 2102 Reynolds Lane, Louisville, Ky., secretary-treasurer.

MARYLAND—Baltimore City Veterinary Medical Association, the second Thursday of each month, September through May (except December), at 9:00 p.m., at the Park Plaza Hotel, Charles and Madison St., Baltimore, Md. Leonard D. Krinsky, 6111 Hartford Rd., Baltimore, Md., secretary.

MICHIGAN—Central Michigan Veterinary Medical Association, the first Wednesday of every month at 7 p.m. Jerry Fries, 2070 E. Main St., Owosso, Mich., secretary.

Mid-State Veterinary Medical Association, the fourth Thursday of each month with the exception of November and December. Robert W. Acton, 4110 Spring Rd., Jackson, Mich.

Saginaw Valley Veterinary Medical Association, the last Wednesday of each month. Alvin R. Conquest, P.O. Box 514, Grand Blanc, Mich., secretary.

Southeastern Michigan Veterinary Medical Association, the fourth Wednesday of every month, September through May. Louis J. Rossoni, 24531 Princeton Ave., Dearborn 8, Mich., secretary.

MISSOURI—Greater St. Louis Veterinary Medical Association, the first Friday of each month (except July and August), at the Coronado Hotel, Lindell Blvd. and Spring Ave., St. Louis, Mo., at 8 p.m. Edwin E. Epstein, 4877 Natural Bridge Ave., St. Louis 15, Mo., secretary.

Kansas City Veterinary Medical Association and Kansas City Small Animal Hospital Association, the third Thursday of each month at the Hotel President, Kansas City, Mo. Robert E. Guilfoil, 18 N. 2nd St., Kansas City 18, Kan., secretary.

NEVADA—Western Nevada Veterinary Society, the first Tuesday of each month. Paul S. Silva, 1170 Airport Road, Reno, Nev., secretary.

NEW JERSEY—Central New Jersey Veterinary Medical Association, the second Thursday of November, January, March, and May at Old Hights Inn, Hightstown, N. J.

David C. Tudor, R.D. 1, Box 284A, Cranbury, N. J., secretary.

Metropolitan New Jersey Veterinary Medical Association, the third Wednesday evening of each month from October through April, except December, at the Irvington House, 925 Springfield Ave., Irvington, N.J. Bernard M. Weiner, 787 Clinton Ave., Newark, N.J., secretary.

Northern New Jersey Veterinary Association, the fourth Tuesday of each month at the Elks Club, Hackensack. James R. Tanzola, Upper Saddle River, N.J., secretary.

Northwest Jersey Veterinary Society, the third Wednesday of every odd month. G. L. Smith, P.O. Box 938, Trenton, N.J., secretary.

South New Jersey Veterinary Medical Association, the fourth Tuesday of each month at the Collmont Diner, Collingswood, N.J. Marvin Rothman, 718 Dwight Ave., Collingswood, N.J., secretary.

NEW MEXICO—Bernalillo County Veterinary Practitioners Association, the third Wednesday of each month, Fez Club, Albuquerque. Donald W. Fitzgerald, 1825 Lomas Blvd., N.E., Albuquerque, N.M., secretary-treasurer.

NEW YORK—New York City, Inc., Veterinary Medical Association of, the first Wednesday of each month at the New York Academy of Sciences, 2 East 63rd St.,

New York City. C. E. DeCamp, 43 West 61st St., New York 23, N. Y., secretary.

Monroe County Veterinary Medical Association, the first Thursday of even-numbered months except August. Irwin Bircher, 50 University Ave., Rochester, N. Y., secretary.

NORTH CAROLINA—Central Carolina Veterinary Medical Association, the second Wednesday of each month at 7:00 p.m. in the O'Henry Hotel, Greensboro. C. G. Sims, 2450 Battleground Ave., Greensboro, N. Car., secretary.

Eastern North Carolina Veterinary Medical Association, the first Friday of each month, time and place specified monthly. Byron H. Brow, Box 453, Goldsboro, N. Car., secretary.

Piedmont Veterinary Medical Association, the last Friday of each month. J. G. Martin, Boone, N. Car., secretary.

Twin Carolinas Veterinary Medical Association, the third Friday of each month at Orange Bowl Restaurant, Rockingham, N. Car., at 7:30 p.m. J. E. Currie, 690 N. Leak St., Southern Pines, N. Car., secretary.

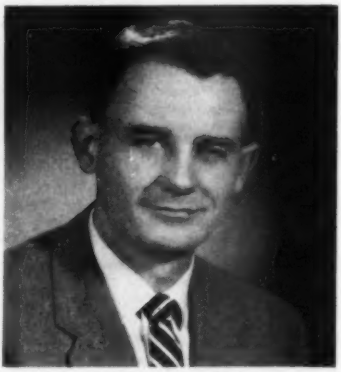
Western North Carolina Veterinary Medical Association, the second Thursday of every month at 7:00 p.m. in the George Vanderbilt Hotel, Asheville, N. Car. Viiu Lind, 346 State St., Marion, N. Car., secretary.

OHIO—Cincinnati Veterinary Medical Association, the third Tuesday of every month at Shuller's Wigwam, 6210 Hamilton Ave., at North Bend Road. G. C. Lewis, 451 E. Galbraith Rd., Cincinnati, Ohio, secretary-treasurer.

Columbus Academy of Veterinary Medicine, every month, September through May. E. M. Simonson, 3120 Valley View Dr., Columbus, Ohio, secretary-treasurer.

Dr. Nicks Named Research Veterinarian for McMillen Feed Mills

Dr. Eugene F. Nicks (GA '55) has been appointed research veterinarian for McMillen Feed Mills, division of Central Soya Co., Fort Wayne, Ind.



Dr. Eugene F. Nicks

After receiving his D.V.M. degree, Dr. Nicks practiced for a year and then was employed by the U.S. Department of Agriculture as area supervisor, veterinary livestock inspector, in North Carolina.

Dr. Edgar R. Marookian Named Merck Specialist

Dr. Edgar R. Marookian (UP '54) has joined the staff of Merck & Co., Inc., Chemical Division, as region technical specialist.

Dr. Marookian will headquarter in Dallas, and his territory will include Texas, Arkansas, southern Illinois, Kansas, New Mexico, and Wyoming.

Since 1954, Dr. Marookian has been in private practice, and has participated in research activities.

Dr. Brinkman Joins Pitman-Moore Staff

Dr. David C. Brinkman has joined the staff of Pitman-Moore Company's veterinary clinical research laboratory, it has been recently announced.

Dr. David C. Brinkman

Born and reared in the state of Washington, he received his D.V.M. from the State College of Washington at Pullman.

Dr. Brinkman was an instructor at the State College for three years and was in private practice for four years. During World War II, he served in the U. S. Army in the Philippines.

Cuyahoga County Veterinary Medical Association, the first Wednesday in September, October, December, February, March, April and May, at 9:00 p.m. at the Carter Hotel, Cleveland, Ohio. F. A. Coy, 8208 Carnegie Ave., Cleveland, Ohio, secretary.

Dayton Veterinary Medical Association, the third Tuesday of every month. O. W. Fallang, 6941 Far Hills Ave., Dayton, secretary.

Killbuck Valley Veterinary Medical Association, the first Wednesday of alternate months beginning with February. C. Gale, Wooster, Ohio, secretary-treasurer.

Mahoning County Veterinary Medical Association, the Fourth Tuesday of each month, at 9:00 p.m. Youngstown Maennerchor Club, Youngstown, Ohio. Sam Segall, 2935 Glenwood Ave., Youngstown, secretary.

Miami Valley Veterinary Medical Association, the first Wednesday of December, March, June, and September. J. M. Westfall, Greenville, Ohio, secretary-treasurer.

North Central Ohio Veterinary Medical Association, the last Wednesday of each month except during the summer. R. W. McClung, Tiffin, Ohio, secretary-treasurer.

Northwestern Ohio Veterinary Medical Association, the last Wednesday of March and July. C. S. Alvanos, 1683 W. Bancroft St., Toledo, Ohio, secretary-treasurer.

Stark County Veterinary Medical Association, the second Tuesday of every month, at McBrides Emerald Lounge, Canton, Ohio. M. L. Willen, 4423 Tuscarawas St., Canton, Ohio, secretary.

Summit County Veterinary Medical Association, the last Tuesday of every month (except June, July, and August), at the Mayflower Hotel, Akron, Ohio. M. L. Scott, 42 W. Market St., Akron, Ohio, secretary-treasurer.

Tri-County Veterinary Medical Association, the fourth Wednesday of January, May, and September. Mrs. R. Slusher, Mason, Ohio, secretary-treasurer.

OKLAHOMA—Oklahoma County Veterinary Medical Association, the second Wednesday of every month, 7:30 p.m., Patrick's Foods Cafe, 1016 N.W. 23rd St., Oklahoma City. Claude A. Tigert, 3032 N.W. 68th St., Oklahoma City, Okla., secretary.

Tulsa Veterinary Medical Association, the third Thursday of each month at the City-County Health Building,

4616 E. 15th St., Tulsa, Okla. Arlen D. Hill, 5302 E. 11th St., Tulsa, Okla., secretary.

Tulsa Association of Small Animal Veterinarians, first and third Mondays, City-County Health Dept. T. E. Messler, 3104 E. 51st St., Tulsa, Okla., secretary.

OREGON—Portland Veterinary Medical Association, the second Tuesday of each month, at 7:30 p.m. Ireland's Restaurant, Lloyds, 718 N.E. 12th Ave., Portland. Donald L. Moyer, 8415 S.E. McLoughlin Blvd., Portland 2, Ore., secretary.

Willamette Veterinary Medical Association, the third Tuesday of each month, except July and August, at the Marion Hotel, Salem. Robert J. Mallorie, P.O. Box 155, Silverton, Ore., secretary.

PENNSYLVANIA—Keystone Veterinary Medical Association, the fourth Wednesday of each month at the University of Pennsylvania School of Veterinary Medicine. Raymond C. Snyder, N.E. Corner 47th St. and Hazel Ave., Philadelphia 43, Pa., secretary.

Lehigh Valley Veterinary Medical Association, the first Thursday of each month. Stewart Rockwell, 10th and Chestnut Sts., Emmaus, Pa., secretary.

Pennsylvania Northern Tier Veterinary Medical Association, the third Wednesday of each odd numbered month. R. L. Michel, Troy, Pa., secretary.

SOUTH CAROLINA—Piedmont Veterinary Medical Association, the third Wednesday of each month at the Fairforest Hotel, Union, S. Car. Worth Lanier, York, S. Car., secretary.

Georgia-Carolina Veterinary Medical Association—see GEORGIA.

TEXAS—Coastal Bend Veterinary Association, the second Wednesday of each month. Jack E. Habluetzel, Route 1, Box 65-N, Ingleside, Texas, secretary.

Dallas County Veterinary Medical Association, the first Tuesday of each month at 7:30 p.m., at a place to be specified. Frank N. Black, 12830 Preston Rd., Dallas, Texas, corresponding secretary.

VIRGINIA—Central Virginia Veterinary Association, the second Thursday of each month at 8:00 p. m., except July and August, at a place in Richmond to be announced monthly. Edwin M. Crawford, secretary-treasurer.

Northern Virginia Veterinary Conference Association, the second Tuesday of each month. T. P. Koudelka, P.O. Box 694, Harrisonburg, Va., secretary.

Northern Virginia Veterinary Society, the second Wednesday of every third month. Meeting place announced by letter. H. C. Newman, Box 145, Merrifield, secretary.

(*Continued on adv. p.* 48)

NEW IMPROVED GLUCOCORTICOID WITH OPTIMAL DURATION OF ACTION

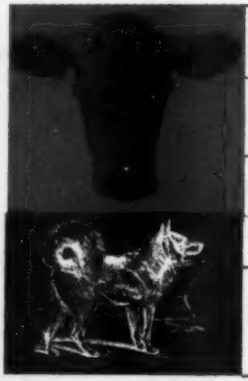
Ultracortenol®

(prednisolone trimethylacetate CIBA)

The result of an intensive search for a longer acting steroid, Ultracortenol exerts greater therapeutic effects following a single injection than other cortisone derivatives.[1] It provides a duration of action that is "just right"—not so short-lived as to produce uncertain results, nor so prolonged as to induce complications (such as undue suppression of lactation).

EASIER AND MORE ECONOMICAL TREATMENT OF SMALL AND LARGE ANIMALS

Ultracortenol has been extensively tested and enthusiastically accepted by a number of small- and large-animal practitioners[1-10] who found the following regimens to be highly beneficial:



DAIRY COWS	
Ketosis (acetonemia)	Single 100- to 200-mg. injection.*
Shock ("downer" cow) syndrome	Single 200-mg. injection as supportive therapy.
DOGS	
Dermatoses	5 mg./10 pounds body weight, total single dose not to exceed 20 mg. For sustained therapy, repeat once or twice a week as indicated.
Inflammatory joint conditions	5 mg./10 pounds body weight, total single dose not to exceed 20 mg. Supportive oral therapy not necessary.

*This initial injection may be reduced to 50 to 100 mg. intramuscularly if simultaneous administration of intravenous glucose is given, thus permitting more economical glucocorticoid therapy. If necessary, either regimen may be augmented by an additional injection of 50 to 100 mg. Ultracortenol after 24 to 48 hours. IMPORTANT: Milk from treated cows should be discarded or used for purposes other than human consumption for at least 24 hours after the last treatment.

SUPPLIED: Multiple-dose Vials, 10 ml., each ml. containing 10 mg. or 25 mg. of prednisolone trimethylacetate in suspension for injection.

ULTRACORTENOL is available from ethical veterinary distributors throughout the United States.

References: 1. Vigue, R. F.: J.A.V.M.A. 133:326 (Sept. 15) 1958. **2.** Shaw, J. C.: Personal communication. **3.** Pollock, S.: Vet. Med. 54:97 (Feb.) 1959. **4.** Rabin, P. H.: Personal communication. **5.** Hoffer, S. H.: Personal communication. **6.** Weir, H. T., and Hazelrig, J. W.: Personal communication. **7.** Beck, J. W.: Personal communication. **8.** Bull, W. S.: Personal communication. **9.** Fessenden, P. E.: Personal communication. **10.** Lohmeyer, C.: Personal communication.

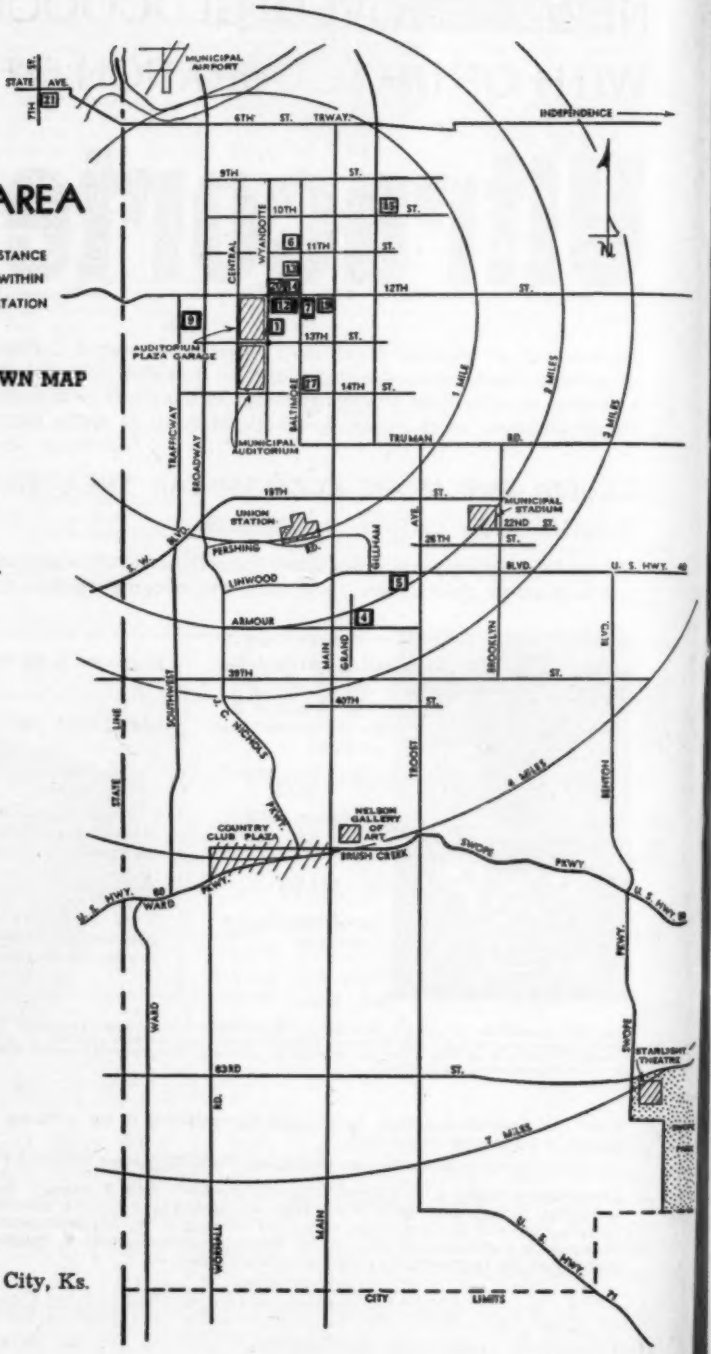
C I B A Effective Veterinary Drugs, Products of Exacting Research
SUMMIT, N. J.

Map of the GREATER KANSAS CITY AREA

ALL HOTELS NOT IN WALKING DISTANCE OF MUNICIPAL AUDITORIUM ARE WITHIN 10 MINUTES BY PUBLIC TRANSPORTATION

HOTEL LIST AND DOWNTOWN MAP

NO.	HOTEL
1	Aladdin, 1213 Wyandotte
4	Bellerive, 214 East Armour Blvd.
5	Berkshire, 1021 E. Linwood
6	Continental, 11th & Baltimore
7	Dixon, 12th & Baltimore
9	Kansas Citian, 1216 Broadway
12	Muehlebach, 12th & Baltimore
13	New Yorker, 1114 Baltimore
14	Phillips, 12th & Baltimore
15	Pickwick, 10th & McGee
17	President, 14th & Baltimore
19	Senator, 17 W. 12th St.
20	State, 12th & Wyandotte
21	Town House, 7th and State, Kansas City, Ks.



HOTEL INFORMATION — KANSAS CITY, MO., CONVENTION

Ninety-Sixth Annual AVMA Meeting, Aug. 23-27, 1959

All requests for hotel accommodations will be handled by a Housing Bureau in cooperation with the Committee on Local Arrangements. The Bureau will clear all requests and confirm reservations.

Hotel and Rate Schedule

Map No.	Hotel	Single	Double	Twin	Suite
1.	Aladdin*	$4.50-8.50	$ 6.50-10.50	$ 9.50-12.00	$17.00-30.00
4.	Bellerive*	5.00-9.00	8.00-12.00	9.00-13.00	From $18.00
5.	Berkshire*	5.00-7.00	7.00-10.00	8.50-10.00	From $14.00
6.	Continental*	6.50-11.00	8.50-13.50	10.00-14.00	$20.00-32.00
7.	Dixon	4.50-7.00	6.50-9.00	8.00-12.00	- - - - - - -
9.	Kansas Citian	3.50-8.00	5.50-11.00	7.00-14.00	From $10.00
12.	Muehlebach*	Headquarters Hotel — No Room Accommodations			
13.	New Yorker	5.50-12.00	8.00-14.00	9.50-14.00	$23.00
14.	Phillips*	7.50-10.50	9.50-13.00	11.50-14.00	$20.50-35.00
15.	Pickwick*	5.85-10.85	6.35-10.85	8.35-12.50	From $14.00
17.	President*	6.50-10.00	9.50-13.00	11.00-15.00	$25.00
19.	Senator	3.50-7.00	5.00-10.00	6.00-10.00	$15.00
20.	State	4.75-6.50	7.50-8.75	8.75-9.25	- - - - - - -
21.	Town House*	5.50-12.00	10.00-13.50	11.00-16.00	From $23.00

*100 per cent air-conditioned; in other hotels listed, majority of rooms air-conditioned.

FAMILY PLAN—The above hotels offer a "family plan" whereby children under 14 years of age will be accommodated in the same room with their parents at no extra charge. If more than one room is required to accommodate children, the hotel will charge only the single rate for each room.

MOTELS—Reservations for motels in the Kansas City area may be made through the Kansas City Convention and Visitors Bureau, 1030 Baltimore Ave., Third Floor, Kansas City 5, Missouri.

PLEASE USE APPLICATION ON REVERSE SIDE FOR HOTEL ACCOMMODATIONS

Application for Hotel Accommodations

1959 AVMA Convention — Kansas City, Missouri

The Convention and Visitors Bureau will make every effort to place you according to your expressed wishes, or to best advantage elsewhere if that is not possible and you desire us to do so.

Please give us the complete information requested below. At least four choices of hotels, or more if you desire, are necessary. Arrange for double occupancy of rooms wherever possible; only a limited number of single rooms is available.

Date.....

Please make hotel reservation in accordance with the following:
Accommodations desired:

Hotel.....First Choice

Hotel.....Second Choice

Hotel.....Third Choice

Hotel.....Fourth Choice

.....Room WITH bath for one person Rate per room desired $..... to $.....

.....Room WITH bath for two persons (double bed) Rate per room desired $..... to $.....

.....Room WITH bath for two persons (twin beds) Rate per room desired $..... to $.....

.....Large room WITH bath for..... persons Rate per room desired $..... to $.....

.....Suite—Parlor, ...Bedroom(s) with bath for ..... persons. Rate per suite desired $.....

Arrival date....., hour.....A. M. .....P. M.
Departure date.....

If reservation cannot be made in one of the hotels indicated shall we place you elsewhere? Yes..... No.....

Please check your mode of transportation: Car..... Train..... Plane..... Bus.....

Rooms will be occupied by (NAMES OF ALL PARTIES MUST BE LISTED)
(PLEASE PRINT)

NAME	STREET ADDRESS	CITY	STATE

.....Check here if you desire accommodations on the FAMILY PLAN.

Name .....

Street address .....

City ..... State .....

Mail to: Convention and Visitors Bureau, 1030 Baltimore Ave., Kansas City 5, Mo.

a complete

vitamin-mineral dietary supplement—with taste appeal to small animals

pet-tabs

This new meat-flavored dietary supplement contains nineteen important ingredients in tablet form. Small animals like them!

The comprehensive Pet-Tabs formula:

- Balances the daily ration.
- Speeds recovery from illness.
- Shortens convalescence after surgery.
- Eliminates complicated additions to rations.
- Maintains health and vigor in older animals.
- Aids growth in young animals.

Dosage: 1 Pet-Tab daily.
Supplied: In bottles of 50. To veterinarians only.

Send for literature

THE S. E. MASSENGILL COMPANY
Veterinary Division
Bristol, Tennessee

Southwestern Virginia Veterinary Medical Association, the first Thursday of each month. D. F. Watson, Blacksburg, secretary.

WASHINGTON—Seattle Veterinary Medical Association, the third Monday of each month, Magnolia American Legion Hall, 2870 32nd W., Seattle. Roy C. Toole, 10415 Main St., Bellevue, secretary.

South Puget Sound Veterinary Association, the second Thursday of each month except July and August. B. D. Benedictson, 3712 Plummer St., Olympia, Wash., secretary.

WEST VIRGINIA—Kyowva (Ky., Ohio, W. Va.) Veterinary Medical Association, the third Thursday of each month in the Hotel Pritchard, Huntington, W. Va., at 8:30 p.m. Harry J. Fallon, 200 5th St., W. Huntington, W. Va., secretary.

WISCONSIN—Central Wisconsin Veterinary Medical Association, the second Tuesday of each quarter (March, June, Sept., Dec.) C. R. Carlson, 1109 E. LaSalle Ave., Barron, Wis., secretary.

Coulee Region Veterinary Medical Association, the third Wednesday of every other month. F. N. Petersen, Box 127, Cashton, Wis., secretary.

Dane County Veterinary Medical Association, the second Thursday of each month. Dr. E. P. Pope, 409 Farley Ave., Madison, Wis., secretary.

Milwaukee Veterinary Medical Association, the third Tuesday of each month, at the Half-Way House, Blue Mound Rd. Dr. Jordan Lewis, Menomonee Falls, Wis., secretary-treasurer.

Northeastern Wisconsin Veterinary Medical Association, the third Wednesday in April. William Madson, 218 E. Washington St., Appleton, Wis., secretary.

Rock Valley Veterinary Medical Association, the first Wednesday of each month. L. C. Allenstein, 209 S. Taft St., Whitewater, Wis., secretary.

Southeastern Veterinary Medical Association, the third Thursday of each month. John R. Curtis, 419 Cook St., Portage, Wis., secretary.

Wisconsin Valley Veterinary Medical Association, the second Tuesday of every other month. John B. Fleming, 209 E. 4th St., Marshfield, Wis., secretary.

Human Cholera in Bangkok—First in 20 Years

In May, 1958, a man died after one day of constant vomiting and diarrhea. In three weeks, there were nearly 5,000 cases; 42 per cent were children. Emergency ambulances, operated by an intern who could give blood transfusions and serum treatments, enabled the mortality to be lowered to a rarely obtained 10 per cent.

Vaccine against the *Vibrio comma* infection was brought by aircraft from all parts of the world and in three weeks nearly 1.5 million people were vaccinated.

Since infection is only by mouth, the disease can be avoided by strict sanitation. Patients were segregated in hospital wards and those who died were immediately cremated.—*World Health (Feb., 1959): 22.*

CLASSIFIED ADVERTISEMENTS

PERSONAL WANT ADS—$4.00 for the first 25 words and 10 cents for each additional word; 35 cents for use of box number.

TOTAL WORD COUNT must include complete box number address (7 words) or personal address line.

COMMERCIAL WANT ADS—$5.00 for the first 25 words, 25 cents for each additional word; $1.00 for use of box number. (See paragraph above for total word count.)

Remittance must accompany ad.

DEADLINES

1st of month issue — 6th of month preceding date of issue.

15th of month issue — 20th of month preceding date of issue.

Names of classified advertisers using key letters can not be supplied. Address your reply to the box number, c/o JOURNAL of the AVMA, 600 S. Michigan Ave., Chicago 5, Ill., and it will be sent to the advertiser.

Wanted—Veterinarians

Veterinarian wanted—active managership, veterinary hospital near Chicago, on partnership basis; no capital. Address Box H 35, JOURNAL of the AVMA.

Assistant veterinarian wanted for small animal practice in western New York state. Opportunity and good pay; apartment available. Address Cole Clinic, 2288 Sheridan Drive, Kenmore 23, N. Y.

THE NEW ZEALAND MEDICAL RESEARCH COUNCIL

HYDATID RESEARCH UNIT

Applications are invited for the post of IMMUNOLOGIST with the Hydatid Research Unit at North Taieri, near Dunedin, New Zealand.

This post is available for a three year period working on immunological aspects of hydatid disease. Previous experience with this disease would be an advantage but other research experience based on a sound knowledge of immunology and pathology would be adequate.

The salary offered for this position is £N.Z. 1800 per annum.

Applications close on *30 June 1959*.

Further particulars may be obtained from the Secretary, Association of Universities of the British Commonwealth, 36 Gordon Square, London, W.C.1. or from the undersigned, C/- University of Otago Medical School, P.O. Box 913, Dunedin, NEW ZEALAND.

C. A. MONROE
Administrative Officer.

Excellent opportunity with Nevada State Department of Agriculture for graduate veterinarian with minimum of two years postgraduate experience in general laboratory diagnosis. Civil service position, salary range: $589-$713. Location, Reno. Duties: laboratory diagnosis, field investigation of animal diseases, and related work. Promotional future for qualified man. Division of Animal Industry, P.O. Box 1209, Reno, Nev.

Veterinarian—willing, capable veterinarian to assist in extensive general practice in Missouri. County Brucellosis regulatory field work is available. State availability, experience, and key information. Must be draft-exempt, neat, nonalcoholic and a credit to community. Salary $400 per month plus $1,000 bonus for first year. Can lead to profit-sharing basis. Will pay one-half of travel expenses for a personal interview. Address Box H 34, JOURNAL of the AVMA.

Assistant in North Carolina veterinary hospital. Small animal practice 90 per cent, large animal practice 10 per cent; 100-cage hospital. Opportunity for a partnership for the right man. Address Box H 43, JOURNAL of the AVMA.

Veterinarian with Master's degree wanted for laboratory and diagnostic work. Address Norden Laboratories, Lincoln, Neb.

Graduating senior or recent graduate for pet hospital in heart of New York City. Excellent opportunity for practical experience. Address Box K 1, JOURNAL of the AVMA.

Wanted—mature associate veterinarian for A.A.H.A. hospital Washington D.C. area. References and curriculum vital in responding letter. Starting salary $7,500. Address Box K 8, JOURNAL of the AVMA.

Veterinarian, Maryland license, for well-equipped hospital, growing small animal practice. Recent graduate acceptable; partnership possibilities. State qualifications. Address Box K 9, JOURNAL of the AVMA.

Veterinarian wanted—assistant in mixed practice, leading to partnership. Virginia license required. Address Box K 14, JOURNAL of the AVMA.

Wanted—Positions

Graduate veterinarian, now in special graduate studies in public health, capable executive and personnel administration, desires challenging position in teaching; laboratory diagnostics; commercial sales, promotion, development; human or animal health program. Address Box H 38, JOURNAL of the AVMA.

High school graduate ('59) wants job as veterinarian's assistant and kennel man. Three years experience working with a mixed practitioner while in high school. Available in June. Address Box K 3, JOURNAL of the AVMA.

Licensed veterinarian with year's experience desires relief work in Texas while awaiting Army call. Available around July first. Address Box K 5, JOURNAL of the AVMA.

Graduate veterinarian desires position in small animal practice in Ohio or Indiana. Would consider lease or eventual purchase. Available upon discharge from Army Veterinary Corps this fall. Address Box K 12, JOURNAL of the AVMA.

Wanted—Practices

Wanted—to buy, or a partnership, in small animal practice in California. Haxe six years experience in Iowa in mixed practice. Graduate of Kansas State College. Address Box H 39, JOURNAL of the AVMA.

Small animal practice, California or Ohio. Thoroughly experienced, married, dependable, excellent references. Have substantial cash to handle. Address Box K 11, JOURNAL of the AVMA.

Seek purchase or lease with option of going small animal practice in middle Atlantic state. Also consider partnership arrangement. Well experienced; approved school. Address Box K 15, JOURNAL of the AVMA.

For Sale or Lease—Practices

For sale—Ohio general practice, established over 20 years, 65 per cent large and 35 per cent small animal practice. Gross for last five years over $45,000. Price includes 11-room house, small animal hospital, drugs, and equipment. Price: $40,000. Details on inquiry, terms can be arranged. Address Box H 7, JOURNAL of the AVMA.

Excellent, zoned, small animal site, Liverpool, New York. Established veterinarian eight miles away will assist new owner to develop practice. Unusual offer. Write Veterinarian, Box 32, North Syracuse, N.Y.

One or two small animal practitioners are desired to lease a rapidly-growing practice on the peninsula, just 25 miles south of San Francisco. Good, separate living quarters. Dr. E. W. Paul, 443 Lakeview Way, Redwood City, Calif.

For sale—mixed practice, mostly cattle and small animals. Includes modern hospital, with outside runs. Also drugs and equipment. Contact Dr. W. W. Adams, Clinton, S. Car.

For sale—Virginia mixed practice. Excellent opportunity for young veterinarian. Low down payment, and balance paid as rent. Address Box K 2, JOURNAL of the AVMA.

Established practice in New York State. Winter months, 70 per cent large animals; summer, resort area, 80 per cent small animals. Sale includes modern home with attached hospital. Priced real estate value only; other interests. Address Box K 4, JOURNAL of the AVMA.

For sale—small animal hospital with living quarters on main street of large Massachusetts city. Write for full details. Address Box K 6, JOURNAL of the AVMA.

For sale—small animal practice. Maryland license required. Address Box K 7, JOURNAL of the AVMA.

For sale—modern small animal hospital and new home, Rocky Mountain area. Excellent opportunity; rapidly growing area. Address Box K 10, JOURNAL of the AVMA.

For sale or lease—modern, fully equipped small animal hospital and practice, suburban Baltimore, Md. Address Box K 13, JOURNAL of the AVMA.

For sale—mixed veterinary practice in central Wisconsin, drugs, and equipment—car and house optional. Reason for selling—health. Address Box K 16, JOURNAL of the AVMA.

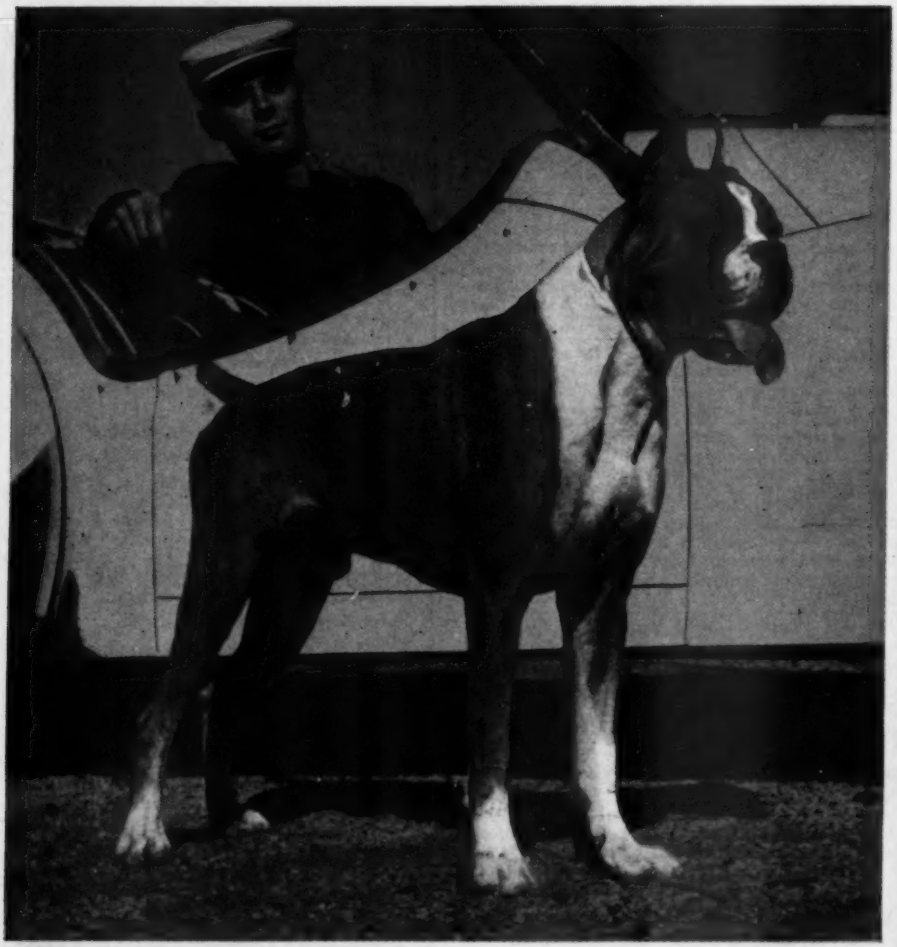
For sale—general practice in central Michigan. Drugs, equipment, and house; $6,500 to handle. Address Box K 17, JOURNAL of the AVMA.

Small animal practice in Baltimore, Md., suburbs for sale. Excellent arrangement for young man with limited capital. Address Box K 18, JOURNAL of the AVMA.

Index to Advertisers in This Issue

THE KEN-L-BISKIT DOG:

healthier... handsomer... stands out from the rest



Costs only PENNIES A DAY to feed him the Dog Food of Champions

Ken-L-Biskit . . . so nutritious, it's the official food at more A.K.C. shows than all other dog foods combined. So dependable, America's top kennels have used it to develop generation after generation of champions. So digestible, even puppies thrive on it. Complete . . . needs no expensive supplements. Get it today, in the thrifty fifty-pound bag.

NOW YOU CAN STOP MASTITI

without building drug resistance or irritating mammary tissue!

STEREOCIN

No other mastitis ointment like it! Here at last is a true veterinary exclusive, a formula that *meets the challenge of changing mastitis etiology* and the most rigid demands of modern dairy scientists.

Consistently bactericidal, STEREOCIN eliminates a major obstacle to mastitis control—*bacterial drug resistance.* The exclusive new antimicrobial, Sterosan®*, is highly effective against streptococci, staphylococci and E. *coli.* In combination with neomycin and bacitracin, Sterosan achieves greatly intensified activity against all common mastitis organisms. There's *no penicillin, no sulfa or streptomycin* in STEREOCIN, and *no irritants* to cause udder damage.

STEREOCIN is supplied in display cartons of 12 individually packaged 10 gram tubes. Acquaint yourself and your dairymen clients with this remarkably advanced mastitis therapy today. With STEREOCIN on hand, you're both equipped *for a better approach to mastitis control.*

Jensen-Salsbery Laboratories, Inc., Kansas City, Missouri

*brand of chlorquinaldol sold under license from Geigy Chemical Corporation

Jen-Sal